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Edinburgh and East of Scotland

College of Agriculture

A Further Contribution to the
Pathology and Epidemiology
of Swine Fever

BY:

J. P. M'GOWAN

M.A., M.D., M.R.C.P.E.

13 GEORGE SQUARE, EDINBURGH

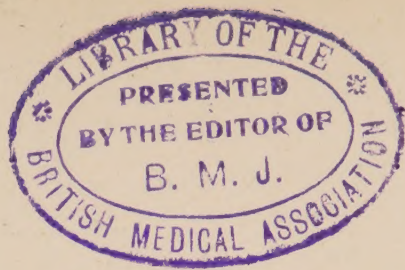
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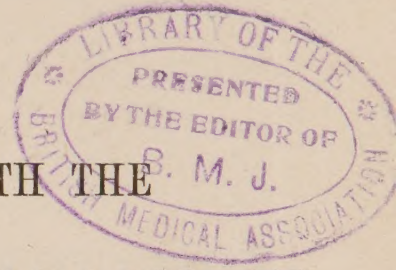
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SOME POINTS IN CONNECTION WITH THE

PATHOLOGY AND EPIDEMIOLOGY OF SWINE FEVER

together with
A FURTHER CONTRIBUTION

BY

J. P. M'GOWAN

M.A., M.D., B.Sc., M.R.C.P.E.

ASSISTANT SUPERINTENDENT, ROYAL COLLEGE OF
PHYSICIANS' LABORATORY, EDINBURGH

*(FROM THE LABORATORY OF THE ROYAL COLLEGE OF
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THE PATHOLOGY AND EPIDEMIOLOGY OF SWINE FEVER.

INTRODUCTION.

Two diseases, swine plague and swine fever or hog cholera, have long been described as occurring in swine. Swine plague, in its septicæmic form, was first differentiated from swine erysipelas by Löffler in 1885 and recognised as an independent disease. Shortly after Schütz (1886) established the ætiological identity of a lung form of the disease with this septicæmic form. Thus, swine plague was characterised by the post-mortem appearances of an acute septicæmia in some cases, in others by pneumonic changes in the lung. Both types of the disease were held to be caused by the *Bacillus suisepiticus*, a member of the hæmorrhagic septicæmia group of organisms. Swine plague was, however, later for a short time, identified by various authorities (Billings, &c.) with hog cholera or swine fever. In America, Salmon (1886) and Smith (1890) separated swine plague sharply from swine fever or hog cholera by the fact that characteristic intestinal lesions were present in cases of the latter disease. It should, however, be noted that these two diseases were recognised to occur together in the same animal very frequently. Salmon and Smith considered that the *Bacillus suispestifer* was the cause of hog cholera or swine fever. It is further to be noted in connection with one point which is dealt with in this paper, that Smith differentiated seven varieties of *B. suispestifer* (a-η).

Subsequent to this, the view has been enunciated (and it is the one current at the present time) that neither *B. suisepiticus* nor *B. suispestifer* are the *primary* causes of diseases in swine.

The primary cause is held to be a filter passer or ultramicroscopic organism, which prepares the way, by lowering the vitality, for a secondary invasion by both or one or other of these organisms. According to the manner of such invasion, it is held, the clinical and pathological varieties of the diseases classed as swine fever are produced.

The main epidemic of swine fever, discussed in this article, occurred during March 1915, in a farm in the vicinity of Edinburgh. The disease was diagnosed as swine fever by the Board of Agriculture authorities. Post mortems were performed by me on eleven cases, and cultures were made from various organs and lesions. The reactions of the various organisms obtained in these cultures are given in Table I. It should be noted in reference to this table that—

- (a) The lung cultures are all from different animals.
- (b) The spleen cultures are all from different animals.
- (c) The gland cultures are all from different animals.
- (d) A lung, a spleen, and a gland culture may all be from one animal.
- (e) That in one organ two different cultures may have been obtained, or two different strains of the same organism. These are usually denoted in the table by the letters α and β , or by certain numerals along with these letters. Nineteen specimens were examined altogether from the eleven cases, and this numeral refers to the number of the specimen in this series.

In discussing this table, the first point that arises is, whether it is possible to group the organisms in any method according to their reactions. Thus it might seem possible, at a first glance, to divide them into organisms with small and organisms with large colonies on slope agar. But against such a classification is the fact that very often a big colony organism was observed to become a small colony organism. Sometimes the cause of this was not evident, but it was definitely observed in several cases that a variation in acid reaction, such as might occur even in one batch of agar, was sufficient to produce it. A gross example of this fact was also witnessed where a batch of agar had accidentally been made too acid. The small colony

TABLE I.

	17.	14 a.	12.	11.	6 b.	8 a.	2 a.	5 a.	4 a.	5 b.	2 b.	8 b.	14 b.	18.	19.	3.	10.	6 a.	4 b.	6 a. Second Strain.	6 a. Third Strain.	9 b.	9 a.	12. Second Strain.	12. Third Strain.	12. Fourth Strain.	15.
SOURCE.	LUNG.	LUNG.	LUNG.	LUNG.	GLAND.	LUNG.	SPLEEN.	SPLEEN.	SPLEEN.	SPLEEN.	SPLEEN.	LUNG.	LUNG.	LUNG.	LUNG.	LUNG.	SPLEEN.	GLAND.	SPLEEN.	GLAND.	GLAND.	LUNG.	LUNG.	LUNG.	LUNG.	LUNG.	LUNG.
Galactose . .	Acid	Acid	O	O	O	O	O	O	Acid	Acid	O	O	Acid	O	O	O	O	Acid	Acid	Acid	? Acid	O	Acid	Acid	O	Acid	? Acid
Maltose . . .	Acid	Acid	O	O	O	O	O	O	Acid	Acid	O	? Acid	Acid	O	O	O	O	O	Acid	O	O	O	O	Acid	O	Acid	O
Glucose . . .	Acid	Acid	O	O	O	O	O	O	Acid	Acid	O	? Acid	Acid	O	O	O	O	Acid	Acid	O	O	Acid	O	Acid	O	O	O
Mannite . . .	O	O	O	O	O	O	O	O	O	Acid	O	Acid	O	O	O	O	O	O	Acid	O	O	O	O	Acid	O	Acid	O
Inuline . . .	Acid	Acid	O	O	O	O	O	O	? Acid	? Acid	O	? Acid	? Acid	O	O	O	O	O	? Acid	O	O	O	O	O	O	Acid	O
Lactose . . .	Acid	Acid	O	O	O	O	O	O	O	? Acid	O	? Acid	Acid	O	O	O	O	O	? Acid	O	O	O	O	O	O	O	O
Dextrine . .	Acid	Acid	Acid	Acid	Acid	O	O	? Acid	Acid	O	O	Acid	Acid	Acid	Acid	Acid	O	Acid	Acid	Acid	? Acid	O	O	? Acid	O	Acid	O
Litmus milk .	Acid	Acid	O	O	O	O	O	O	O	O	O	O	Acid + clot	Acid + clot	Acid	Acid	O	O	O	O	O	O	O	O	O	Acid + clot	O
Raffinose . .	O	Acid	O	O	O	O	O	O	O	O	O	O	Acid	O	O	O	O	O	Acid	O	O	O	O	O	O	O	O
Potato . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Salicine . . .	O	O	O	O	O	O	O	O	O	O	O	? Acid	Acid	O	O	O	O	O	Acid	O	O	O	O	O	O	O	O
Sorbite . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	Acid	O	O	O	O	O	O	O	O	O
Dulcite . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Cane	Acid	Acid	? Acid	O	O	O	O	O	O	O	O	? Acid	Acid	O	Acid	Acid	O	O	Acid	O	O	O	O	O	O	Acid	O
Adonite . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Peptone W. .	no Indol	no Indol	Indol present	Indol present	Indol present	Indol present	O	O	no Indol	no Indol	O	O	Indol present	no Indol	no Indol	Indol present	O	Indol present	no Indol	no Indol	O	O	Indol present	no Indol	O	no Indol	present
Nitrites . . .	marked production	O	O	marked production	slight	marked production	slight production	marked production	marked production	? production	marked production	slight production	? production	? production	? production	? production	O	O	marked production	? production	O	? production	? production	produced	O	marked production	marked
Gelatine . . .	no growth	no growth	no growth	no growth	no growth	no growth	slight growth	slight growth	slight growth	slight growth	slight growth	slight growth	no growth	no growth	no growth	no growth	no growth	slight growth	slight growth	slight growth	very slight growth	heavy growth	no growth	heavy growth	growth	slight growth	no growth
Agar	small colony	small colony	large colony	large colony	large colony	small colony	small colony	small colony	small colony	large colony	large colony	large colony	large colony	small colony	small colony	large colony	large colony	small colony	large colony	small colony	small colony	large colony	small colony	large colony	large colony	large colony	large colony
Guinea-pig (in-traperitoneal)	recovered : marked pus, no bacteria in films	recovered : pus, no bacteria in films	not tested	dead : large amount clear exudate, little pus, cocco-bacilli, polar staining	dead : large amount clear exudate, little pus, polar staining, cocco-bacilli	Ill: pus, polar staining, cocco-bacilli	dead : clear exudate, little pus, bipolar bacilli	alive : pus, no bacteria in films	alive : pus, no bacteria in films	alive : pus, no bacteria in films	alive : pus, no bacteria in films	alive : pus, no bacteria in films	dead : pus, bipolar cocco-bacilli	alive : pus, no bacteria in films	alive : pus, no bacteria in films	dead : clear exudate, little pus, bipolar cocco-bacilli	dead : clear exudate, little pus, bipolar cocco-bacilli	dead : clear exudate, little pus, bipolar cocco-bacilli	alive : pus cells filled with bipolar cocco-bacilli	alive : pus cells no bacteria in films	alive : pus, no bacteria in films	alive : pus, no bacteria in films	not done	alive : pus, no bacteria in films	dead : clear exudate, little pus, bipolar cocco-bacilli	dead : clear exudate, little pus, bipolar cocco-bacilli	
Voges and Proskauer	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Motile	non	non	non	non	non	non	non	non	non	non	non	non	? one or two bacilli motile	non	non	non	non	non	non	non	non	non	non	non	non	non	non
Gram	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative	negative
Morphology according to Scheme α^*	C	C	A	A	A	E	D	B	B	B	B	B	A	C	C	B	B	B	E	F	F	F	C	F	C	C	C
Morphology according to Scheme β^+	II.	II.	II.	II.	II.	II.	I.	I.	I.	I.	I.	I.	II.	II.	II.	I.	I.	I.	II.	I.	I.	I.	II.	I.	II.	II.	II.
Appearance in original culture	pure culture small colonies	mixture of small and large colonies	pure culture large colony	pure culture large colony	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	pure culture small colonies	pure culture small colonies	pure culture large colonies	pure growth of large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	mixture of small and large colonies	pure culture large colony	pure culture small colony	pure culture large colony	pure culture large colony	pure culture large colony	pure culture large colony

* Scheme α —A = hardly visible coccal forms, short bacilli, and debris (may be a variety of C).
B = great difference in size small cocci, large cocci, bipolar, and girdle forms.

C = minute cocco-bacillus in chains.
D = large cocco-bacillus varying in size (may be a variety of B).

E = minute coccus or cocco-bacillus with staphylococcal groupings (may be a variety of C).
F = fat short cocco-bacillus all same size—polar staining (may be a variety of B).

† Scheme β —I. = great difference in size small cocci, large cocci, bipolar, and girdle forms.
II. = minute cocco-bacillus.

organisms died out on this medium, while the large colony organisms either died or became very small. It is characteristic, too, of organisms of the hæmorrhagic septicæmia group, that they grow best on a medium with a definite alkaline reaction. In such a case the small colony organisms approached in the size of their growth the large colony ones. It should be noted further, that both types of organism had a resemblance to one another, in that both tended to die out at times on different media in an unaccountable fashion. From the table, too, it will be seen that often an organism, recorded as a large colony one, gave the reactions—to mention only one, growth on gelatine—of a small colonied one and *vice versa*.

The following, quoted from Uhlenhuth and Haendel's article on Swine Fever (1), has a bearing on this point (p. 388). They state that the *Bacillus suispestifer* produces mutation forms on agar giving rise to different types of colony. Thus in one strain there appear on the agar plate two types of colony—an opaque large filamentous one, together with a small, clear, smooth-edged one; while in another strain, in similar circumstances, there appear clear transparent colonies and dull opaque ones; while in a third strain, one obtains clear transparent ones along with small opaque mother-of-pearl-like colonies. Such colonies also show differences in their morphological appearances microscopically, yet it is to be noted that they are all considered to be the same organism.

It would appear, therefore, to be impossible to divide these organisms according to the size of the colonies as exhibited on agar, but as a working basis, for ease of discussion, this method of division will be temporarily adhered to.

The next point to be considered is, whether this division into small and large colonies holds for the microscopical appearances of the organisms in the table. Taking the simpler scheme of division according to microscopic characters in the table—the division into varieties α and β —it will be seen that large colony organisms give both varieties of microscopic appearances and small colony organisms do the same. It should be noted, however, that all the organisms are cocco-bacilli. All the organisms again of both types are gram negative and non-motile with the possible exception of 14 b, which may be slightly motile.

With regard to the pathogenicity to guinea-pigs when injected intraperitoneally, both large and small colonies failed to kill guinea-pigs in certain cases, and in regard to both when the guinea-pig in such cases was killed, the findings in the peritoneum could be the same—namely, the presence of numerous pus cells and the entire absence of bacteria, although a large number of these had been injected. When, on the other hand, as occurred frequently, the intraperitoneal injection of either type killed the guinea-pig, the findings in both could again be the same—namely, a large amount of clear exudate, swarming with bacteria and with practically no leucocytes. In both cases the organisms present in the fluid were typical bipolar bacilli.

As regards the growth on stab gelatine, none of them produced liquefaction, and, as mentioned above, sometimes a large colony produced hardly any growth in this medium, while, on the other hand, it sometimes happened that a small colony produced a good growth.

As regards the reactions of these various organisms on the various sugars, &c., and other media, it can only be said that in this respect no attempt at a general law can be formulated. The reactions of the large colonies on the sugars no more resemble one another than they resemble the reactions of the small colonies. The same may be said also of the small colonies. The only possible generalisation that might be made is, that this very erratic behaviour of members of the two groups constitutes one of their greatest resemblances to each other.

The general facts, therefore, derivable from a consideration of Table I. are:—

- (1) All the organisms are bipolar cocco-bacilli.
- (2) All were obtained from typical cases of swine fever.
- (3) They were obtained either as pure original cultures of small and large colonies, or as mixtures of these. No other organisms except these two were obtained in any of the cultures.
- (4) One of the greatest resemblances between the members of the two groups is their capacity for variation.
- (5) These two groups shade one into the other.

The small colonies undoubtedly represent *Bacillus suisepiticus*, and the question comes to be, what is the nature of the large colonies? They may be—

- (1) *B. suisepiticus* on a favourable medium such as agar of an optimum alkalinity.
- (2) Looked at from the side of *Bacillus suispestifer* they may be aberrant forms of *B. suispestifer*.

With regard to this latter point, the following quotations are apposite.

JOEST (2) states that BÖDER found that the fermentative powers of *Bacillus suispestifer* are independent of the amount of sugar present and of the reaction of the medium. They are, however, dependent on the temperature of incubation, on the kind of medium (apart from the fermentable substance), and on the particular virulence of the culture. The Preisz strain of *Bacillus suispestifer*, unless in a virulent condition, caused only slight acid but no gas production in glucose media. This strain, after it had been passed through a series of guinea-pigs, produced both acid and gas in glucose media. Preisz and Karlinski found that the strains of *Bacillus suispestifer* known to them produced no gas in glucose.

BAINBRIDGE (3) states that the paratyphoid group of organisms are all motile, gram negative, do not form indol, are cocco-bacilli with a tendency to bipolar staining, do not liquefy gelatine, and while fermenting glucose, mannite, dulcitol, lævulose, galactose, maltose, and arabinose with acid and gas production, produce no apparent change in lactose, cane, raffinose, salicin, and inulin. He then mentions that a striking exception to the above statement was met with. One strain of *Bacillus suispestifer* never formed gas in any carbohydrate medium, although it produced acid in the same media as the other strain of this bacillus. This observation was repeated several times at intervals of some weeks always with the same result. In other respects the two strains were indistinguishable from one another, and presumably, he states, the one strain while retaining its power of forming acid from various carbohydrates had never possessed or had lost the capacity to form gas. He mentions that a similar strain had been described by Bock in 1906.

Uhlenhuth and Haendel (*op. cit.*, p. 387) mention that *Bacillus suispestifer* will grow at 45°C., but by so doing it loses its motility and ceases to form gas in glucose. They mention further (p. 389) that certain strains of *Bacillus suispestifer* do not pro-

duce gas in glucose, and also that certain strains may produce acid and gas in lactose.

Uhlenhuth and Haendel, p. 396, obtained eight species of *B. suipestifer* from hog cholera cases. They resembled this organism very closely, but did not agglutinate with a hog cholera serum. One called *Paratyphoid C.* culturally was the same as *B. suipestifer*, but did not agglutinate with *B. suipestifer* serum. Other strains culturally resembling this *Paratyphoid C.* strain would not agglutinate with a *Paratyphoid C.* serum. Sobernheim and Seligman also found many strains resembling *B. suipestifer* culturally which would not agglutinate with *Paratyphoid B.* serum, nor with *B. suipestifer* serum.

Sobernheim and Seligman, Haendel and Gildmeister, &c., show that under certain conditions this *Paratyphoid B.* group can vary in their cultural and serological reactions so much that the original character is entirely changed. In many cases Haendel and Gildmeister obtained from swine fever cases a large number of strains which culturally behaved like *B. suipestifer*, but which agglutinated neither with *B. suipestifer* serum nor with *B. Gaertner* serum. They were all agglutinated with serum prepared by the injection of one of themselves, and also agglutinated with serum prepared with Gläser and Voldagsen strains. Certain of these strains agglutinated at a later period in their history with a *B. suipestifer* serum, while others after three years did not do so. Sera prepared later from some of these strains agglutinated *B. suipestifer* up to the limit of the titre, but did not at all agglutinate *B. Gaertner*. They did not, however, show any change in their original agglutination reaction to one another, or to Gläser or Voldagsen strains.

Gläser's *Bacillus typhi suis* and the *Bacillus suipestifer* Voldagsen which have just been mentioned are the cause of two contagious diseases in pigs resembling swine fever. Culturally and serologically they differ very little from each other. In all probability they are both varieties of *B. suipestifer*. Milk is usually unaltered, but in some cases the reaction on this medium is the same as with *B. suipestifer*. There is very great variety in their actions on glucose media. Sometimes gas is formed, and sometimes not. The same culture may produce it at one time, and not at another.

A spontaneous infection of healthy pigs occurs when these healthy pigs are housed with pigs artificially infected with these organisms (Gläser, Dammann and Stedefeder, Pfeiler). Uhlenhuth and Haendel agree that, as with *B. suispestifer*, this may occur especially in young animals, but that in these diseases there is not the high infectivity of natural swine fever.

It should be further mentioned that Dr Wang and I (4) have shown that it is possible to increase the biological activities of the *Bacillus avisepticus* by *passage* through animals. Thus an organism which originally fermented some sugars with acid formation only, is by this procedure rendered capable of fermenting them and others with acid and gas production. In working on chicken cholera, and sheep and cattle septicæmia, I have met numerous examples of such enhanced organisms in natural disease conditions. (5) (6).

Taking everything into consideration, therefore, a feasible explanation of all the phenomena seems possible only by considering that all the organisms met with in Table I. are variants of *Bacillus suissepticus*. So far as we have gone, however, we have not brought forward any cogent evidence that, looking at it from the standpoint of *Bacillus suispestifer*, this organism may be a variant of *Bacillus suissepticus*. It is true that I have adduced evidence from the literature that *Bacillus suispestifer* may vary within wide limits, even to resembling closely the organisms classed as large colony ones in Table I. It is necessary in this connection, therefore, to consider the organisms obtained from the organs of a single case of another outbreak of swine fever. These are given in Table II.

It should be noted here that no other organisms except those belonging to the two groups, small clear and large whitish, were obtained from any of the organs in this case. Cultures were made from the lung, glands, and intestine. Small colonies resembling those described in Table I., as evidenced by their reactions, were obtained from the three situations. Large colonies obtained from a gland, intestine, and lung were also tested on the media, and while it was found that the organisms from the gland and intestine compared in their reactions with the large colonies of Table I., the organism from the lung differed markedly, resembling in many respects a culture of *B. suispestifer* obtained

TABLE II.

SOURCE.	1.		2.		3.		4.		5.		6.		7.		8.	
	Gland.	Intestine.	Intestine.	Lung.	Lung.	Intestine.	Lung.	Intestine.	Lung.	Intestine.	Lung.	Intestine.	Gland.			
Galactose	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	Acid	•	•	•	Acid	
Maltose	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	•	•	•	•	•	
Glucose	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	•	•	•	•	Acid	
Mannite	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	Acid	•	•	•	Acid	
Inuline	•	•	•	•	[O]	•	•	•	•	•	•	•	•	•	•	
Lactose	•	•	•	•	[O]	•	•	•	•	•	•	•	•	•	•	
Dextrine	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	•	•	•	•	Acid	
Lact. N. R.	•	•	•	Fluorescence	[Fluor.]	•	•	•	•	•	•	•	•	•	•	
Litmus milk	•	•	•	? acid	[alkaline]	•	•	•	•	•	•	•	•	•	•	
Raffinose	•	•	•	•	[O]	•	•	•	•	•	•	•	•	•	•	
Potato	•	•	•	snail track	[?]	•	•	•	•	•	•	•	•	•	•	
Salicine	•	•	•	•	[O]	•	•	•	•	•	•	•	•	•	•	
Sorbite	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	•	•	•	•	•	
Dulcite	•	•	•	Acid + gas	[A + G]	•	•	•	•	•	•	•	•	•	•	
Cane	•	•	•	Acid	[O]	•	•	•	•	•	•	•	•	•	•	
Adonite	•	•	•	•	[O]	•	•	•	•	•	•	•	•	•	•	
Peptone W.	•	•	•	no Indol	[none]	•	•	•	•	•	•	•	•	•	•	
Nitrites	•	•	•	no nitrites	[+]	•	•	•	•	•	•	•	•	•	Indol present marked pro- trites	
Gelatine	•	•	•	good growth	[+]	•	•	•	•	•	•	•	•	•	no growth	
Agar	•	•	•	large colony	...	•	•	•	•	•	•	•	•	•	small colony	
Guinea-pig inoculation	•	•	•	not done	...	•	•	•	•	•	•	•	•	•	not done	
Voges and Prosk.	•	•	•	negative	[negative]	•	•	•	•	•	•	•	•	•	negative	
Motile	•	•	•	non	[non]	•	•	•	•	•	•	•	•	•	non	
Gram	•	•	•	negative	[neg.]	•	•	•	•	•	•	•	•	•	negative	
Morphology	•	•	•	large cocco- bacillus	...	•	•	•	•	•	•	•	•	•	small coccus or cocco-bacillus	
Appearances in original culture	•	•	•	large cocco- bacillus	...	•	•	•	•	•	•	•	•	•	2 small clear colonies	
Colony examined	•	•	•	30 small clear colonies, 3 large white colonies	...	•	•	•	•	•	•	•	•	•	small colony	

Swine fever case: the lower end of the small intestine, part of cæcum, with necrotic and chronic healing ulcers; bronchial glands; and piece of old-standing chronic pneumonia with pleural adhesions all from one case.

Intestinal cultures showed: Culture 1, 40 large white colonies, 40 small clear colonies; culture 2, 100 large white colonies, 40 small clear colonies; culture 3, 100 large white colonies, 20 small clear colonies.

Lung cultures showed: Culture 1, 100 small clear colonies; culture 2, 100 small clear colonies, 19 large white colonies; culture 3, 30 small clear colonies, 3 large white colonies.

Gland cultures showed: Culture 1, 1 small clear colony, 1 large white colony; culture 2, 6 small clear colonies, 6 large white colonies; culture 3, 2 small clear colonies.

from Wassermann, the reactions of which are placed alongside it in Table II. Thus in this one case there exist three organisms resembling one another in basal facts, but one of them differing from the other two in the reactions on the various sugars, and resembling closely, but being not quite the same as, Wassermann's strain of *B. suispestifer*. It should be further specially noted that organism 2, Table II., a large colony, *obtained from the intestine*, gave very weak reactions in the various sugars, &c.

Taking everything into account, therefore, it would seem that evidence has been adduced for the very great probability that *B. suispestifer* is only an enhanced variety of *B. suissepticus*. To complete the material in hand relative to the variations in the organisms found in swine fever, the reactions of a group of organisms obtained from the organs of a pig dead of swine fever, from a third outbreak, are given in Table III. Culture 5, whose reactions are also given in this table, was a stock culture whose origin was unknown.

It should be noted that only two organisms—the large and the small colony—were obtained from the organs in this case; and that while the bronchial glands and the heart-blood gave pure cultures of the large colony, pure extensive cultures of the small colony were obtained from eight different parts of the lungs. The reactions of the large colonies here, it should be observed, are not characterised by any marked action on the sugars.

In summarising this section it may be stated:—

- (1) In cases from three epidemics of swine fever, diagnosed as such by the Board of Agriculture authorities, organisms roughly divisible into a small colony type and a large colony type were obtained.
- (2) These two types of organisms were the only organisms obtained from the various organs and lesions of these cases.
- (3) The reactions of the large and small type of colony on the various media would appear to be of a similar nature. Differences do markedly occur, but these are not in basal characters. In fact, a point of great similarity between the two types is this very capacity for varying. This variation is further along the same lines in the two types, and intermediate forms exist.

TABLE III.

	1.	2.	3.	4.	5.
SOURCE.	LUNG.	LUNG.	BRONCHIAL GLAND.	HEART-BLOOD.	(Stock culture.)
Galactose . .	O	O	O	O	Acid
Maltose . .	O	O	O	O	O
Glucose . .	O	O	O	O	Acid
Mannite . .	O	O	O	O	O
Inuline . .	O	O	O	O	Acid
Lactose . .	O	O	O	O	O
Dextrine . .	O	O	O	O	O
Lact. N. Red	O	O	O	O	O
Litmus milk .	O	Acid ?	O	O	Acid
Raffinose . .	O	O	O	O	O
Potato . . .	O	O	O	O	O
Salicine . .	O	O	O	O	O
Sorbite . .	O	O	O	O	Acid
Cane . . .	O	O	O	O	Acid
Adonite . .	O	O	O	O	...
Peptone W. .	Indol	Indol	Indol	Indol	Indol
Nitrites . .	trace	trace	trace	trace	trace
Gelatine . .	no growth	no growth	no growth	no growth	no growth
Agar . . .	small colony	small colony	large colony	large colony	small colony
Guinea-pig in- oculation	not done	died in 24 hours	not done	not done	not done
Voges and Proskauer	negative	negative	negative	negative	negative
Motile . . .	non	non	non	non	non
Gram . . .	negative	negative	negative	negative	negative
Morphology .	Polar staining cocco-bacillus	Polar staining cocco-bacillus	Polar staining cocco-bacillus	Polar staining cocco-bacillus	Polar staining cocco-bacillus
Appearances in original culture	Pure extensive culture, small colonies	Pure extensive culture, small colonies	Pure extensive culture, large colonies	Pure culture, 22 colonies of large colony	...

Pig died of swine fever ; several large ecchymoses on abdomen and behind ears, fat and in good condition ; no peritonitis, large and small intestine covered with petechiæ. Mesenteric glands greatly enlarged ; lungs, old-standing pneumonia, with pus in bronchi ; bronchial glands very large and congested. Small intestine shows intense congestion, with petechiæ in its wall, and covered with tenacious muco pus ; at places hæmorrhage into the lumen of the gut. No growth of any kind was obtained from the mesenteric glands or spleen.

Bronchial glands: Culture 1, pure culture of numerous fairly large greyish colonies (reactions 3) ; culture 2 shows same.

Heart-blood: Culture 1, 6 large whitish-grey colonies as in bronchial glands ; culture 2, 22 large whitish-grey colonies as in bronchial glands (reactions, see 4).

Lungs: 8 cultures were made from 4 different parts of the lungs, and each gave a pure extensive culture of a small colony (reactions 1 and 2).

- (4) The view is therefore expressed here, that in all probability, so far as microscopically visible organisms are concerned, we are dealing with only one organism and its variants, the *B. suis* *septicus*, a member of the hæmorrhagic septicæmia group, a group well recognised to be capable of great variation in virulence, morphology, and biological activity on the part of its members.

HISTORY OF THE EPIDEMIC THE REACTION OF WHOSE ORGANISMS ARE DETAILED IN TABLE I.

The disease occurred in a piggery in connection with a large institution near Edinburgh. The outbreak commenced about the beginning of March. All ages were affected. The first symptom noted was that the pigs appeared to be going off their food. After this had lasted for about a week, acute cases began to occur, especially among the old sows and boars. Such animals would be refusing their food one day, be much worse the next day, and dead the next. The temperature in one of these cases was 110° F. Further, in these rapidly fatal cases a watery diarrhœa was present.

As regards general symptoms in the rest of the cases, there was hardly any discoloration behind the ear or in the axilla or groin; no coughing was observed in any of the cases; only in two cases in this epidemic was there running at the eyes. Emaciation took place after the animals had been ill for some time; two sucking pigs died after having had fits, one of them after a very prolonged fit. Some of the pigs appeared paralysed, and lay and kicked on the floor until they had produced large abrasions over bony prominences. Diarrhœa in these more chronic cases appeared as a late symptom in the disease.

STATE OF THE PIGS BEFORE THIS EPIDEMIC.

Previous to the epidemic the pigs had been doing very well indeed. There were, however, one or two "wasters" among the animals, and these had been present for the last six months. In connection with this, it should be mentioned that

the pigman had often noticed that even in apparently healthy pigs, the slightest exertion caused them to cough. He looked on this as quite normal. It should further be noted that epidemics of diarrhoea among sucklings at three weeks old, and again at six weeks, were a common occurrence in this piggery. The further fact should be noted that the pigman, a man who had twenty years' experience in this piggery, and had gone through four previous epidemics, stated that the epidemics in his experience occurred mostly in February. Only one epidemic had occurred remote from this time, when it happened in July: he regarded February as the dangerous month.

METHOD OF FEEDING, HOUSING, ETC., OF THE PIGS.

The pigs are fed on refuse of human food from the institution; this is all cooked. No food is brought in from outside except "sharps" obtained from a large grain-dealer in town. The food is given warm in winter, and the pigman has often noticed that the pigs shivered after eating it. The pigs are kept in separate small pens, one part of it roofed over, the other part an open court. The floors are of concrete. There is no artificial heating; the pens are cleaned out every day and fresh straw supplied.

PREVIOUS EPIDEMICS.

As previously mentioned, the pigman has been in his present situation for twenty years, and has had experience of four previous epidemics.

As regards the present epidemic, he cannot account for it in any way. The piggery was thoroughly disinfected after the last epidemic three and a half years ago, and a fresh new stock brought in. Since this took place there has been no introduction of fresh stock, with the exception of a boar which was brought in two years ago.

The epidemic immediately previous to this one took place as stated three and a half years ago in July. This epidemic was said to be due to the lifting of old floors and the laying of new ones, infection presumably having hung about the place from a previous epidemic.

The epidemic previous to this last occurred about four or five years earlier, and was attributed to a man and cart from the place attending a market where a large number of pigs affected with swine fever were being sold, while the epidemic before that was attributed to farmyard manure being brought on to the fields from a place where swine fever had occurred.

The pigs were affected differently in different epidemics. Thus in one epidemic a very prominent symptom was that the pigs stood and bored their heads and noses into a corner of the house. They also coughed a good deal, and subcutaneous hæmorrhages were markedly present. In another epidemic running at the eyes was very prominent, and was the first symptom noted. The eyes were as red "as those of a Russian rabbit." There was also running at the nose. In some epidemics he noticed a considerable number of the pigs were paralysed in their hindquarters. They dragged them, and appeared weak in the back. Again, cases where they lay kicking on the floor until they were covered with abrasions were quite frequent.

POST MORTEM FINDINGS.

CASE 1. Shott: died from the disease; showed subperitoneal petechial hæmorrhages on the small intestine; no lesions in the mucosa of intestine. Petechial hæmorrhages on the pericardium; bronchial glands and abdominal glands enlarged, red, and hæmorrhagic. Anterior part of every lobe of the lung showed old-standing consolidation with pus exuding from the bronchi. This tissue was tough, grey in colour, and resembled on section a piece of pancreas.

CASE 2. Shott: very ill; killed; petechial hæmorrhages in spleen; no intestinal lesions; abdominal lymphatic glands enlarged and congested; condition of thoracic organs same as in Case 1.

CASE 3. Shott: alive; so ill that it cannot stand; with kicking on the floor it had produced large excoriations on the outside of the right foreleg; petechial hæmorrhages were present in the spleen. Abdominal glands were enlarged and red. No intestinal lesions were present. Petechiæ were present in the heart subepicardially. There was acute consolidation of the anterior parts of all the lobes of the lung. They were deep red

and looked like blood clot. The middle lobe on the right side was specially affected. The bronchial glands were enlarged and congested.

CASE 4. Same conditions as in Case 3.

CASE 5. This had been a "waster" for the last six months. It was not apparently very ill at the present time. It was killed, and on examination showed hæmorrhages into the spleen and on to the outer surface of the colon and cæcum. There were no characteristic intestinal lesions. The spleen was not enlarged (there was no enlargement of the spleen in any of the cases).

On opening the thorax, the lungs were found to be adherent to the ribs and to the pericardium. All the lobes were more or less solidified; there were minute hæmorrhages all over them, and behind the right lung was a large mass of inspissated pus in the pleural cavity; the surface of the pericardium was covered with hæmorrhages; pericarditis was not present, nor were there any hæmorrhages in the epicardium. The bronchial glands were enlarged, congested, and red, as were also those in the root of the neck.

CASE 6. Same history and same condition post mortem as in Case 5.

CASE 7. Same condition as in Case 3.

CASE 8. Same condition as in Case 3.

CASE 9. Old sow, very acute case, died in three days from onset of illness. Nothing seen post mortem except enlarged congested lymphatic glands, petechial hæmorrhages in various parts, and congestion of the lungs.

CASE 10. Old sow, very acute case, dead in three days. Same condition found post mortem as in sow 9, with the addition of several large hæmorrhages beneath the mucous membrane of the stomach.

CASE 11. Shott: killed. Same condition found as in Case 3, with the addition of several typical ulcers in the large intestine.

In several of the cases it has to be further noted that there was present a considerable effusion into the peritoneal cavity.

The main points in connection with these post-mortem examinations may be summarised thus:—

In the very acute cases little was observed except widespread petechial hæmorrhages, enlargement and congestion of lymphatic glands, congestion of the lungs, and hæmorrhages, often large, in the stomach wall.

With regard to the more chronic cases, discoloration of the skin was practically absent from all the cases, petechial hæmorrhages were observed in various situations, the spleen was not enlarged in any of the cases, only one case showed the definite typical intestinal lesions, the mesenteric and other lymphatic glands were enlarged, congested; and hæmorrhagic.

With regard to the lesions in the lungs, some points need to be emphasised. While in many of the cases there was evidence of recent disease, in the majority of the cases there was also evidence of old-standing trouble. There were patches of old, grey, tough, solid, bloodless, pancreas-looking tissue, of a date much antecedent to the observed outbreak of the disease.¹ In two cases especially (Cases 5 and 6) there was also evidence of an old-standing empyema. In these two cases there was further the definite history of "the animals have been ill for a long period." It should be stated further, that from the bloodless patches of grey solidified lung, organisms of the kind mentioned in Table I. were obtained. It need only be further mentioned that the organisms whose reactions are detailed in Table I. were derived from the organs of the cases whose pathological conditions have just been detailed.

Several important points in connection with the epidemiology of swine fever may now be discussed with reference to this outbreak. As already mentioned, no cause from the outside could be assigned for its occurrence. Limiting ourselves, however, for the present to a consideration of the internal facts of the case, we have the following accepted facts to begin with.

That the organisms *Bacillus suispestifer* and *suisepcticus* can live a saprophytic life outside the body of swine, in the earth,

¹ In two of the epidemics referred to in this paper, the swine not obviously affected with the disease and apparently in good health were removed to the slaughter-house and killed there, with the object of using them for human food provided they turned out not to be diseased. I had opportunities of seeing the internal organs of a considerable number of these animals, and in a very large number of them these grey tough patches were present in the lungs.

dung, &c. It is true that their virulence when existing in such circumstances is not, however, great.

That the same organisms can live a saprophytic-parasitic existence in the respiratory passages and alimentary tract of apparently healthy swine. It may be granted, too, that in such conditions they are comparatively avirulent.

It is further recognised that such organisms, especially *B. suis*, become virulent and capable of producing fatal disease when from some condition the vitality of the animal is lowered. As a corollary to this, it is well recognised that hæmorrhagic septicæmia may occur either as sporadic cases or widespread epidemics.

There is evidence in this case that many of the pigs had been suffering from disease definitely attributable to *B. suis* for some time prior to the outbreak of the disease. Such evidence consists in the old-standing lesions, due to this organism, found in the lungs of some of the cases, and the definite history in two of the cases of a long-standing illness in addition.

The occurrence of a large number of cases at one time does not necessarily mean that this is due to infection from one case to the other. It can be quite well explained by the view that these cases occurred simultaneously owing to a single extensive secondary cause acting on all the pigs together.

In this connection it should be borne in mind—

- (1) That chronic cases of the disease were occurring in all probability previous to the disease.
- (2) That March weather is very inclement, and in this instance was especially so.
- (3) While the pigs were undoubtedly housed, their courts, to which they had access, were quite open to the elements.
- (4) While the pigs were housed in separate pens, the disease was occurring in all the pens at the same time.
- (5) In the report of the Chief Veterinary Officer of the Board of Agriculture for the year 1909, p. 31, it is stated that “we have several times placed healthy pigs in styes left untouched for three days, fourteen days, and thirty days after they had been vacated by undoubtedly affected pigs, but in no case have we been able in this way to produce a clear case of swine fever.”

Considerations such as these lead me to the position that, while the possible contagious nature of swine fever is not denied,¹ there is no cogent evidence for accepting it as explaining either the initial outbreak nor its continuance in this case. We have the statement recorded above of the experience of the Chief Veterinary Officer as to the actual infectivity of swine fever. Yet in this case there is no record of anything at all approaching such gross exposure; indeed, there is no record of the pigs having been exposed to infection at all. The disease would appear, therefore, not to have come from a fully developed case outside, and no pig had been introduced for at least two years. There was, however, evidence of some of the pigs having old-standing lesions such as are well known to be caused by organisms of the hæmorrhagic septicæmia group, apart altogether from the assumption, warranted from the amount of careful work directed towards this point, that such organisms could be already present in the respiratory passages, &c., of the animals, and in the manure, earth, &c., of the sty (7).

I have already shown (8) that braxy in the sheep is due to an invasion by the *Bacillus ovisepticus* of the sheep consequent on a lowering of vitality due to gross chilling. And I see no reason why such an assumption should not equally explain this outbreak of numerous *sporadic* cases (the evidence only takes us so far), more especially as several of the animals were already suffering from old-standing lesions due to the *Bacillus suisæpticus*.

What has been said of the nature of the infectivity in this case applies equally to the statements made with regard to original outbreaks of the disease. As mentioned above, no explanation could be found for the origin of this last epidemic, and far-fetched explanations, as already recorded, were offered for the earlier outbreaks in this piggery. In this connection I quote again from the Chief Veterinary Officer's report for

¹ To explain the epidemiological facts, the following may be considered as a possibility. By the operation of secondary causes, an ordinarily avirulent strain may become virulent, and while in this condition may be capable of acting as a sole cause. A condition of this kind probably occurs in human pneumonia. Here the pneumococcus is found in the mouth, &c., of large numbers of healthy individuals. In the spring months the operation of a secondary cause, namely, cold, originates sporadic cases of disease, but it is known that occasionally direct infection occurs from such cases to healthy individuals in which the secondary causes have not been operative.

1909, p. 30: "In many cases, I think I may say in the majority, one establishes by careful tracing some direct or indirect connection between one outbreak and another. It may be a sow sent to the boar on premises where swine fever was afterwards found to exist; it may be the direct purchase of pigs from infected premises on which an undeclared outbreak exists or from a market which has been infected by such pigs; it may be through a travelling castrator. In quite a number of cases, however, the direct connection with a well-established outbreak dates far beyond the incubation period, three or four or even six or eight months. *Faute de mieux* one is compelled to accept the explanation that swine fever may lurk for months on premises without giving any marked indication of its existence." This statement should be compared with the extract already given above from the same article (p. 22).

The underlying assumption in all such views is that a case of the disease must necessarily arise from a previous definite case of the disease. In the case under discussion there was no evidence of any such direct transmission. The position, however, may be better summed up by saying that were swine fever as infectious as fulfilment of the theories usually advanced for new outbreaks would necessitate it to be, then there would exist no piggeries free from the disease, and such experimental results as have been recorded in the report of the Chief Veterinary Officer already mentioned (*v.* p. 22) would be utterly impossible. There is also the fact that many pig proprietors, by careful attention to the general health of their swine, after a long experience have never had the disease among their stock, even when their habit is to buy pigs in the open market for feeding.

Emphasis has been laid up to the present on the possible explanation as a sporadic occurrence of an epidemic diagnosed by competent authorities as typical swine fever. Nothing has been said as yet with regard to the cause of swine fever in general, except that in this instance certain bacterial findings in the cases are tested as to whether or not they offer a reasonable explanation of the phenomena as observed. The question of the possible contagiousness of the various diseases resembling the so-called swine fever has now to be considered.

CONTAGIOUSNESS OF SWINE PLAGUE, &C.

Ostertag (quoted by Uhlenhuth, p. 336) holds the view that in Germany a contagious disease of swine caused by the *Bacillus suisepcticus*—true or classical swine plague—does occur as a separate entity, affects old animals as well as young, and can cause a mortality of anything up to 75 per cent. He also maintains that a further disease—*chronic* swine plague of pigs—is directly connected with this *acute* destructive classical swine plague. The acute form may pass into the chronic, and as such may again introduce the acute fatal form, and with the material of both forms either of the forms may be alternately produced.

Hutyra (quoted by Uhlenhuth, p. 335), however, believes that, in swine plague as in swine fever, the primary cause of the disease is a filter passer, and that the clinical characters of these two diseases are determined by a secondary invasion of *Bacillus suisepcticus* or *suipestifer*. He admits the possible occurrence of the classical pure swine plague as sporadic cases or as an enzootic, but not as an epizootic. With regard to the chronic swine plague in sucklings, he doubts if it is derived from the classical swine plague just mentioned, and considers that the *Bacillus suisepcticus* alone is not responsible for its occurrence. Uhlenhuth and Haendel (p. 336) do not deny the possible occasional occurrence of classical swine plague. They, however, think, like Hutyra, that the pneumonia frequently observed in swine fever cases is in most cases a sequel to the infection with the swine fever virus (filtrable virus), and is not of the nature of a superadded infection with a second contagious epidemic disease, swine plague. They think that the lung changes can be caused by the filtrable virus alone, but that often other bacteria such as *Bacillus suisepcticus*, normally present in the respiratory passages of healthy swine, come into play.

With regard to a possible contagious disease attributable to *Bacillus suipestifer*, Uhlenhuth and Haendel (p. 330) mention that Schreiber insists that *Bacillus suipestifer* is the cause of the contagious disease swine fever. Schreiber does not question the possible production of swine fever with filtered material from a previous case, for he himself has produced it in this

way. He holds, however, that the filtrable virus is only the toxin of *Bacillus suipestifer*, and that one can only produce swine fever by injection of such filtered material when *Bacillus suipestifer* is already present somewhere in the body of the experimental animal.

Uhlenhuth and Haendel (p. 333) admit that there is a disease of swine attributable to the *Bacillus suipestifer* which resembles swine fever very closely. This organism occurs very abundantly in the organs of such cases, and by being intravenously or intrastomachically administered it can cause a disease resembling swine fever both clinically and pathologically. They, however, state that this disease is quite different from swine fever, and that the same changes can be produced by the injection of other organisms of the *coli* group and also by dead cultures of *Bacillus suipestifer*.

Gläser (quoted by Uhlenhuth, p. 334) states that while he admits the occurrence of the "filter passer" disease, there exist two other infectious diseases of swine of almost equal contagiousness. One of these is caused by a bacillus resembling the human typhoid bacillus, the other by *Bacillus suipestifer*.

Dammann and Stedefeder state that there are two different ætiological entities included under the name swine fever. One of these is caused by the "filtrable virus," while the other is caused by an organism, the *Bacillus suipestifer* Voldagsen.

Pfeiler also thinks that these organisms are capable of causing a contagious disease among swine. Uhlenhuth and Haendel (p. 334) themselves obtained the *Bacillus suipestifer* Voldagsen on two occasions from epidemics of so-called swine fever. They considered, however, that the disease caused by this organism, *though contagious*, was not so contagious as swine fever. Even in this case, however, an attempt is made to show that the organism is a secondary invader on the top of a filtrable virus infection. They state that they found the "filtrable virus" in one case but not in the other. It should further be noted, however, that they admit that they can produce the disease with the bacillus alone, and that when it is so produced it is contagious. This admission should be borne in mind, because later it will be seen that one of the points of distinction which Uhlenhuth and Haendel draw between the disease produced by the filter passer and by the

Bacillus suispestifer is, that the first is contagious and the other is not.

It cannot be denied, therefore, that on reviewing the evidence a case is made out for the existence of definite contagious diseases simulating closely swine fever, one caused by *B. suis-septicus* and the other by *B. suispestifer* and its varieties. (*B. Voldagsen* and *typhi suis* are regarded as variants of *suispestifer*.)

As regards the contagious disease caused by *B. suis-septicus*, it will be noted that while it may cause sporadic cases and enzootics among adults, it is chiefly mentioned as causing a virulent disease with a mortality of 50-80 per cent (Ostertag) among young pigs and sucklings. I (6) have already drawn attention to the fact that, in hæmorrhagic septicæmia of the sheep, while there is not much evidence in some epidemics of contagiousness of the disease among adults, there is evidence that the disease is readily transferable from the mother to the lambs sucking her, in the form of "white scour." On the natural pasture, where sheep graze widely, there is comparatively little opportunity for lambs infecting one another, and as a matter of fact there is no evidence that they do.

Again, in connection with chicken cholera, I (5) have shown that there is a great deal of evidence that this disease can exist sporadically, enzootically, and epizootically among old fowls, but that among young chicks it occurs as a virulent contagious epidemic in the form of white diarrhœa. Again, in the case of calves, I have adduced evidence that white scour, a very contagious disease, is in all probability the contagious analogue of sporadic and enzootic hæmorrhagic septicæmia in cattle. It should be noted that in both these last cases—the chicks and the calves—the young animals are closely herded together, and that thus opportunities of infection are much greater than among lambs.

The pigman who had charge of the pigs implicated in the epidemic here investigated stated that the only two diseases he had to contend with among his swine were swine fever and diarrhœa in young pigs at three weeks and six weeks old. This diarrhœa appeared to him very infectious, and would affect all the members of a litter. It often left behind it "piners" or "wasters," and a report of the post-mortem findings in two such wasters is given above in cases 5 and 6. In these cases

pure cultures of *B. suis* were obtained from the lesions in the lungs. No opportunity has occurred as yet, however, of investigating the acute diarrhoea of the pigs, but the resemblance of the clinical facts of this case to those in the calves, chicks, and lambs, deserves to be noted.

I come now to a more definite discussion of the possible rôle of the filter passer in swine fever. Before discussing it directly, however, some general points bearing on the methods of diagnosis in use with regard to swine fever deserve attention. In dealing with these I am again quoting from Uhlenhuth and Haendel's paper.

They state (p. 364) that very often there is great difficulty in establishing with certainty a diagnosis of swine fever. All serological methods of diagnosis ordinarily in use for other diseases leave us in the lurch here.

Owing to the extraordinary variety of clinical symptoms in the disease picture, there is great difficulty in establishing a diagnosis. One can only suspect the disease from them.

Again, the definite diagnosis of the disease from the pathological findings in slaughtered or dead animals is difficult, because the pathologico-anatomical findings vary so enormously. *Where at the post mortem one finds the characteristic appearances in the intestine, such as diphtheritic inflammation with necrosis and scurfing and the flat well-known ulcers, the diagnosis can be taken for certain, even if in some cases these changes are the result of a bacterial infection such as has been long known to be caused by the B. suis, B. enteritidis (Gaertner), certain coli-like organisms found by Uhlenhuth, &c., the Bacillus typhi suis, and the Bacillus suispestifer Voldagen.*

If, on the other hand, such bacteria are found, it cannot be concluded on this account that the disease in question is not true swine fever.

Occasionally, as has been mentioned, gross organic changes are not present. Thus the characteristic appearances, inflammation of the intestinal mucosa and ulcers, may be entirely absent. In these cases lung changes, especially the necrotic pneumonia, as was once held typical for swine plague, are the only demonstrable findings. In such cases, because similar lung changes occur in pure swine plague, caused solely by the *B. suis*, the determination of whether we have to do with

pure swine plague or with true swine fever is a matter of great difficulty. *Even after Bacillus suisepiticus has been cultivated from the lung lesions, and the absence of intestinal lesions has been demonstrated in such a case, the diagnosis of swine plague cannot be made without something further being done. The epidemiological evidence must be examined and the post-mortem findings in other cases in the epidemic must be considered before a diagnosis is made. If you find changes in the intestinal canal of some of the other animals, then you can conclude that you are dealing with swine fever.*

Further inoculation experiments with filtered material from the cases may be tried, *but it should be noted that while a positive result is of value, a negative result does not mean that the disease is not swine fever.*

It is difficult to make an adequate précis of such diagnostic hints. The gist of the matter, however, seems to be, that if one finds intestinal lesions in a case—lesions which, as Uhlenhuth admits, can equally well be produced by a great variety of other organisms (including killed *B. suispestifer*), and which others have shown to be present in other contagious epidemic diseases of swine definitely proved to be caused by organisms of the *suispestifer* group (a fact which Uhlenhuth and Haendel admit)—then you are entitled to give a diagnosis of swine fever. If you are in doubt in any particular epidemic, owing to the non-existence of these lesions, you examine numerous cases until you find them. The diagnosis is then clinched if you find such a case. If, after having examined numerous cases, you find no intestinal lesions, you may filter the blood, &c., and inject an experimental pig. This animal may not take the disease. You are not, however, to conclude from that that the disease is not true swine fever. The epidemiological evidence may help. If the disease appears to be contagious, or better, very contagious, the diagnosis is swine fever. A standard of contagiousness, however, is not mentioned, nor is it stated that many of the diseases from which you have to diagnose swine fever, such as *suispestifer* Voldagsen disease and swine typhoid, are contagious as well,—some state as contagious as true swine fever.

In short, and to be perfectly frank, the diagnostic methods given here are grossly biassed. They are designed not for an

impartial consideration of the case, but in order to forward a preconceived theory of the cause of the disease. The result of their use is, apart from the errors arising from grossly erroneous statements such as have been pointed out with regard to the diagnostic use to be made of intestinal lesions and contagiousness, that a position is arrived at where, if an observation favours in any degree the filtrable virus view, it is accepted and no cognisance whatever is taken of other circumstances which emphatically point in another direction.

If, therefore, such methods of reasoning are to be used in the routine examination and diagnosis of swine fever, what results, one asks, are likely to be obtained when such methods are applied, as they have been, to experimental work—say, for instance, the determination as to whether or not a pig has died of true swine fever subsequent to the injection of the so-called filtrable virus. It is rather to be presumed that in such a case it would be argued that, as the material injected—the filtrate—ought from the theory to produce swine fever, then cases so injected, resembling clinically and pathologically and in all other respects the diseases just mentioned, would be separated from these cases and regarded as pure swine fever by this very fact of their having been injected with the filtrate. The whole subject of the investigation and diagnosis of swine fever would appear to be in the grip of a colossal vicious circle—results got by fallacious methods of diagnosis being used to bolster up quasi-experimental results and *vice versa*.

So far the diagnostic methods in vogue have been discussed. Some of the experimental methods in use for the establishment of the filter passer theory will now be examined. In this connection the first point to be dealt with is the method used for the selection of experimental animals.

Uhlenhuth and Haendel (p. 351) state that they selected their experimental animals from an “absolutely spotless piggery.” Pigs must not be bought for such purposes in the open market, for owing to the widespread and often latent course of swine fever, it is all too probable that amongst the pigs collected from everywhere and anywhere by the buyers, and brought to the market, animals already infected may occur. If one should use animals from a doubtful source, they should only

be experimented on after they have been in quarantine *for at least fourteen days*.

The question comes to be, in the first instance, what is to be considered an "absolutely spotless piggery." The pigs in the piggery which was the seat of the epidemic of swine fever dealt with in this paper (*vide supra*), a week previous to the outbreak of the epidemic, were, in the words of the pigman, "thriving and doing exceptionally well." No swine fever had been in the piggery for the past two and a half years, and there was no evidence whatever of outside infection. Yet a week after swine fever broke out and 200 pigs were slaughtered on this account.

Again Uhlenhuth and Haendel mention that it is inadvisable to buy pigs in the open market owing to the *widespread and often latent course of swine fever*, yet they state that such pigs (with latent swine fever in them possibly) may be used for experimental purposes *after a quarantine period of fourteen days*, possibly in an experiment for the determination of whether a so-called virus filtrate is capable of producing swine fever.

The question is further complicated if we consider the possibility of the existence in pigs to be used for experiment of latent or chronic lesions of the other diseases liable to be confounded with swine fever, as for instance such conditions as chronic swine plague, Voldagsen disease, or swine typhoid. The difficulty of a diagnosis has already been discussed, and if pigs have to be selected, as undoubtedly they must be, free from all traces of these diseases as well as of swine fever, the difficulty of selection becomes much greater, and the sources which one can draw from much circumscribed.

Again, the possibility cannot be excluded that even the presence in the body of pigs of organisms of the groups mentioned, *B. suis* *septicus*, &c., is a source of danger to the reliability of an experiment and may vitiate it. It has not been disproved that the so-called filtrable virus may possibly be a toxin derived by the action of the blood serum on such bacteria. Toxins derived in this manner, by digesting such organisms with rabbit serum and subsequently filtering them, have been shown by me to produce hæmorrhages in various parts of the body, especially the intestine of the rabbit, subsequent to injection. It is not supposing too much to put

forward the view that such hæmorrhages, if produced in the pig's intestine, could be infected by the *B. suispestifer* already present there. Indeed, Uhlenhuth and Haendel state that they have produced all the pathological appearances of swine fever, including the intestinal lesions, by injecting pigs with *dead* cultures of *B. suispestifer*. It might be opportune at this stage, therefore, to point out that the difficulties in the path of the direct proof by experimental injection of pigs that the virus filtrate is the cause of swine fever appear to be insuperable, and that the main evidence for the view that this is the case is wholly of this nature.

It might appear, therefore, that little of advantage is likely to result from a further discussion of the subject, especially with regard to the characters of this virus filtrate, whose very existence appears to be founded on fallacious reasoning. The possibility exists, however, that the disease, swine fever, may be caused by the virus filtrate in spite of the methods by which attention has been focussed on its possible existence. With this in view, it therefore behoves me to consider some of the more important characters which have been attributed to it. At the same time I must point out that, while so doing, I reserve the right to reject any statement bearing on these characters on the general grounds already adduced. For this reason I propose to deal with the subject rather from the point of view of whether these statements made regarding the virus filtrate are not explicable on other grounds than that the virus filtrate is a living multiplying virus. By adopting this method it may happen that some of the statements regarding the characters of the virus filtrate do not lend themselves to reasonable explanation in this manner. In such a case I take the liberty of doubting such statements on the general grounds mentioned above, the onus of rendering them reasonably credible on these grounds, in my opinion, having rested with those making the allegations.

With these considerations in view, therefore, I proceed to deal with the question of some of the characters of virus filtrate. Uhlenhuth and Haendel (page 326 *et seq.*) state that Dorset, Bolton, and M'Bryde first promulgated the idea that the disease was caused by a filter passer in 1905. Ostertag, working later with a Potz filter on cases in Germany, could not

confirm these results, and concluded that the disease as it existed in Germany was different from the American variety. Koske, using a Pukall filter, obtained similar results to Oster-tag. Since that time, however, it is stated that the American results have been confirmed in England, South Africa, Hungary, France, Italy, &c.

Ostertag, working at a later period in conjunction with Stadie, and using serum which he had obtained from American cases, was able to produce the disease. He also tried the method again with German swine fever cases, and obtained five positive results out of eight.

These results, as regards the production of the disease by the filtered material, are not unequivocal, and it has further to be borne in mind in this connection that Uhlenhuth and Haendel, in their recommendations for diagnosis, have found it necessary to state that while a positive result following injection is to be relied on, a negative result does not mean that the disease in question is not swine fever. Further, the frequent occurrence in articles by supporters of the virus filtrate theory of possible explanations as to why negative results are obtained subsequent to injection, is significant of the need for such explanations.

The following considerations are also adduced by some as evidence of the ætiological relationship of a filter passer to swine fever. M'Clintock, Boxmeyer, and Siffer, as well as Theiler (quoted by Uhlenhuth and Haendel, p. 327-28), could not find *B. suispestifer* present in undoubted cases of swine fever. The inference is that, as it was not found, it could not, therefore, be the cause of the disease. It must, however, be stated that neither in these experiments, nor indeed anywhere in the literature, has the third possible factor in the disease—the *Bacillus suissepticus*—been considered as a possible claimant to recognition. The contest has always existed between the upholders of the virus filtrate theory and those who regard *B. suispestifer* and its variants as causal.

The next point to be considered is, granting that the filter passer is the cause of swine fever, is it of the nature of a living multiplying virus? Several investigators (quoted by Uhlenhuth, p. 337) have attempted by various means to enhance the virulence of the filtrate by cultivation outside the body.

It may be stated, however, that with few exceptions—and these not regarded by their authors as conclusive—all the methods have failed. Uhlenhuth quotes a case where he had a result which might be construed in this sense; and later Pfeiler and Lentz (9), 1914, state that they have obtained a multiplication of the virus.

Methods of concentration by means of filtration and centrifuging have failed to bring about their object.

The reaction of the filtrate to various chemical agencies is not of the nature one expects to find in a living virus, but is more in harmony with what one finds in a toxin. Uhlenhuth mentions (p. 332) that in an experiment, while 2.5 per cent antiformin solution killed in thirty to forty minutes *B. suispestifer*, which had been artificially added to serum filtrate, the specific injurious action of the serum filtrate from swine fever, swine under the same conditions was first destroyed after two hours. It must, however, be pointed out that with regard to *B. suispestifer*, the test is growth of the organism on artificial media, while the test for the serum filtrate is injection into swine; and Uhlenhuth and Haendel have already admitted that they could produce the clinical and pathological appearances of swine fever by the injection of *dead* *suispestifer* bacilli. Later they state that the antiformin destroys also the toxin of *B. suispestifer*. On general grounds, however, having in view the very many incontrollable factors in the case, such a statement must be accepted with great reserve.

The next question to be considered is the technique of the preparation of the filtered virus in its relation to the results claimed for it afterwards. Uhlenhuth and Haendel (p. 340) state that in their work various filters have been tried, but those chiefly used were Berkefeld's Nos. 12 and 15. This variety was used because it possessed very small pores. In this connection, however, not only has size of pores to be taken into account, but also possible flaws and cracks in the candles. The work of Bulloch and Craw relative to the reliability of the Berkefeld filters is worthy of consideration in this respect (10). They found that as far as 90 per cent of Berkefeld filters gave a contaminated filtrate within fifteen minutes from the commencement of filtration. M'Bryde (11) also found that *B. suispestifer* can pass through some of the Berkefeld filters.

The sterility of the resultant filtrate was tested generally by adding to the filtering material the *Bacillus fluorescens*. The object of this procedure was to test whether an organism of presumably the same size, namely, *Bacillus suispestifer*, was kept back. The choice of *Bacillus fluorescens* would also undoubtedly be determined by the fact that the fluorescent change produced by it in cultures would signal its presence to the naked eye. The use, however, of such a signal lends colour to the presumption that in many cases the absence of the fluorescence would be regarded as indicating, without further test, that the filtrate was bacterially sterile. Uhlenhuth and Haendel, it is true, give an elaborate statement of the further precautions necessary to ensure the sterility of the filtrate. They mention that bacterial growth may take place in the filtrate without any marked change as regards turbidity being observed. The final test, however, even after all these precautions, is just this appearance or non-appearance of turbidity in liquid media. In this connection the very slender growth of certain strains of *B. suissepticus*, and consequent absence of turbidity in fluid media, especially if they are at all acid in reaction, may be pointed out. It may be further noted that the precautions just mentioned are not directed in any sense specifically towards the exclusion of *B. suissepticus* passing into the filtrate. This is a grave omission, taking into consideration the great variations in size, especially the very minute forms possible in the case of this organism. It has further to be mentioned that many of the organisms of the hæmorrhagic septicæmia group are of such high virulence that a very few organisms are capable of causing disease and death. Thus Voges (quoted by Huttyra (12)) states that he so increased the virulence of *B. suissepticus* by *passage* that, whereas previous to this intensification, 4 mgms. of organisms were necessary to kill a guinea-pig in thirty hours, after it 0.1 milligramme promptly killed a like animal in 3-4 hours, and in certain cases 5-10 bacteria were sufficient to cause death.

Lourens (quoted by Uhlenhuth, p. 330) states, moreover, as his opinion, that *B. suispestifer* breaks up into granules which may pass through a filter and remain in the filtrate without producing turbidity. His experiments are not conclusive, but

in this connection the marked capacity of organisms of the hæmorrhagic septicæmic group to break up into granules, especially when in contact with blood serum, should be noted.

Uhlenhuth and Haendel (p. 331) have advanced the view that the small amount of filtrate necessary to produce the disease by subcutaneous injection is evidence in support of the filter passer theory. Apart altogether from the results of Voges' experiments which have just been quoted as to the very small number of *B. suis* necessary sometimes to produce disease and death, quite another explanation can be found. It is well known that the injection of very small doses of tuberculin, in cases with a latent tuberculous focus, may set up a recrudescence of the disease, and may even give rise to acute miliary tuberculosis. In the same way the injection of the filtrate, even in small doses, may light up old foci of disease through the agency of a specific toxin which it may contain. Once these chronic foci are stirred up, the way is open for a general infection.

Uhlenhuth and Haendel state (p. 333) that while they do not deny that a disease resembling swine fever clinically and pathologically can be produced by the administration of cultures of *B. suis*, this disease is different from swine fever in the following respects:—

- (1) It is not contagious.
- (2) Filtered blood serum from such cases is not infectious.
- (3) After recovery from this disease there is no immunity from infection by natural swine fever.

With regard to No. 1, it has been abundantly shown by Gläser, Dammann and Stedefeder, Pfeiler (Uhlenhuth, p. 395 *et seq.*), as also by Pfeiler and Standfuss, Pfeiler and Kohlstock (9), that *B. suis* and its variants may cause a *very contagious* disease among swine.

With regard to statement 2, Uhlenhuth and Haendel's diagnostic recommendation relative to positive and negative results subsequent to injection of filtered virus should be borne in mind, and with regard to statement 3, it should be remembered that such authorities as Joest and Koske and Preisz (quoted by Uhlenhuth and Haendel, p. 366) hold that the recovery from true swine fever is not in any sense a protection from a second attack.

Granted that the serum filtrate from a case of true swine fever may produce swine fever, can one explain this occurrence in another way than by the assumption that the virus is living and capable of multiplication. Uhlenhuth (p. 333) has, as previously mentioned, produced "pseudo-swine fever"—i.e., a fever resembling swine fever clinically and pathologically—by the injection of killed cultures of *B. suispestifer*. As dead *suispestifer* bacilli were injected in this case, such injection must have given rise to an infection with an organism already present in the body, which organism was capable of producing lesions typical of swine fever. I have further shown that a toxin derivable from *B. suispestifer*, *suissepticus*, &c., on extracting these with fresh rabbit's serum, produces, when injected into rabbits, as one of its most marked pathological changes, hæmorrhages in various parts of the body, especially in the intestine and the vermiform appendix. These might presumably get infected with organisms already present in the intestine. The further fact has to be borne in mind that in rabbits the organism of pseudo-tuberculosis, which in all probability belongs to the hæmorrhagic septicæmia group, produces, when injected intravenously, the symptoms and post-mortem appearances (as just described) of members of this group. Similar effects are seen when this organism causes disease through natural channels of infection. One of the chief lesions found post mortem in this disease is necrotic patches, small and numerous, in the vermiform appendix—that is, in the organ where hæmorrhages are most frequently produced by the toxin of the organisms of the hæmorrhagic septicæmia group. Apart from these considerations, what has been said anent the possible analogy between what may be in operation here and what takes place with tuberculin injections in cases with chronic tuberculous foci, may be repeated in this connection.

Uhlenhuth and Haendel (p. 331) mention that the filtered serum from swine rendered ill by the injection of *B. suispestifer* is not capable of causing infection. It has, however, to be remembered that failures to produce the disease occur frequently when filtered serum from a natural case of swine fever is injected, and that the number of experiments where the *suispestifer* injection has been tried must be only a small fraction of the number where the swine fever filtrate has been

tested. Thus, whereas in the latter case more numerous experiments have tended to show that the injection fails in a good percentage of cases, a more extensive series of experiments in the first case might bring to light a definite positive result. This is indeed probable in the light of Uhlenhuth's own experiments with dead *suipestifer* bacilli mentioned above. It is quite possible, further, that the toxin, if it is a toxin, circulating in the blood of true swine fever cases, is not a toxin derived from *B. suipestifer* alone, but it may also be derived from several variants of this organism, as also from *B. suisepcticus*, and in these matters circumstances of apparently little moment are often of the very greatest importance. With regard to the immunity produced by injecting pigs (especially pigs which have passed through the natural disease) with so-called virulent serum, it is well known in all vaccine work that the nearer the antigen approaches that occurring naturally in the disease, the better will the results of immunisation be. As has just been mentioned, it may be supposed that the antigen manufactured by the action of the serum not only on the *B. suipestifer* but also on the *B. suisepcticus*, will have more immunising virtue than cultures on artificial media of *B. suipestifer* alone. It will not avail anything to enter here into a further discussion of the immunity phenomena of this disease, for the results obtained are specially liable to the operation of the fallacies of diagnosis, discussed at some length in an earlier part of this paper.

CONCLUSIONS.

1. From investigations conducted on material obtained from three epidemics of swine fever, the view is enunciated here that the *Bacillus suipestifer* may vary considerably in its biological reactions, and may even be considered to be a variant of *B. suisepcticus*.

2. From the epidemiological facts observed in connection with one of these outbreaks, the opinion is expressed here—

1. That swine fever may arise in a piggery without any connection being traceable between this outbreak and a previous existing case of the disease outside.

2. That in some epidemics, as the one in question, the evidence is not sufficient for the view being held that in all cases the disease, once having arisen in a piggery, spreads by *contagion* to the other animals in the piggery. It may do so, but it is possible in some cases that contagion may be simulated by some common injurious factor acting on all the pigs simultaneously and producing the disease in them at or about the same time.
3. It is not, however, denied that the disease, once having arisen in a piggery, may spread thereafter through the piggery by contagion.

3. From the examination of this outbreak, and from the knowledge of the numerous cases in the literature, where obvious contagion not being traceable it is assumed that contagion *must have taken place* in some recondite fashion from some previous case, the view is put forward here that swine fever is by no means so contagious as it is usually assumed to be.

4. While the possibility of the filter passer being the cause of swine fever is not denied, the view is expressed here that the evidence for this being the case is far from compelling. It should be specially pointed out that while in the establishment of the causal relationship of the filtrate *B. suispestifer* has been considered, no attention has been paid to *B. suissepticus* in this connection.

RECOMMENDATIONS.

Much could possibly be done, in the light of the results obtained in this investigation, to reduce loss from swine fever by—

1. Attending to the diarrhoea in sucklings.
2. Killing at once, on the first evidence of the condition being present, all “wasters” and “piners” of any degree whatever.
3. Attending more closely to the general health of pigs. Thus, to mention one circumstance, they should be protected from sudden exposure, especially to great cold. For this reason pigs should be kept in pens in large airy houses entirely under cover during the winter season.

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A FURTHER CONTRIBUTION TO THE
PATHOLOGY AND EPIDEMIOLOGY
OF SWINE FEVER

NOTE.

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A. M'CALLUM,

COLLEGE OF AGRICULTURE,
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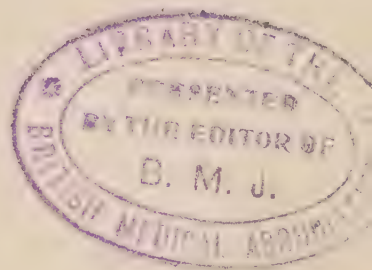
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Edinburgh and East of Scotland College of Agriculture

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BY

J. P. M'GOWAN

M.A., M.D., M.R.C.P.E.



(FROM THE LABORATORY OF THE ROYAL COLLEGE
OF PHYSICIANS, EDINBURGH)

WILLIAM BLACKWOOD AND SONS
EDINBURGH



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A FURTHER CONTRIBUTION TO THE PATHOLOGY AND EPIDEMIOLOGY OF SWINE FEVER.

IN a previous paper, published in July 1915,¹ I dealt with the literature on the subject of swine fever, more especially with relation to the "filter passer" aspect of the subject. It is therefore unnecessary to enter again at length into the matter. It will serve the purposes of this paper if the conclusions and recommendations advanced in that paper are here set down. They were as follows:—

CONCLUSIONS.

1. From investigations conducted on material obtained from three epidemics of swine fever, the view is enunciated that the *Bacillus suispestifer* may vary considerably in its biological reactions, and may even be considered to be a variant of *B. suis* *septicus*.

2. From the epidemiological facts observed in connection with one of these outbreaks, the opinion is expressed—

- (1) That swine fever may arise in a piggery without any connection being traceable between this outbreak and a previous case of the disease outside.
- (2) That in some epidemics, as the one in question, the evidence is not sufficient for the view being held that in all cases the disease, once having arisen in a piggery, spreads by *contagion* to the other animals

¹ Bulletin of Edinburgh and East of Scotland Agricultural College, July 1915.

in the piggery. It may do so, but it is possible in some cases that contagion may be simulated by some common injurious factor acting on all the pigs simultaneously, and producing the disease in them at or about the same time.

- (3) It is not, however, denied that the disease, once having arisen in a piggery, may spread thereafter through the piggery by contagion.

3. From the examination of this outbreak, and from the knowledge of the numerous cases in the literature, where obvious contagion not being traceable, it is assumed that contagion *must have taken place* in some recondite fashion from a previous case, the view is put forward that swine fever is by no means so contagious as it is usually assumed to be.

4. While the possibility of the filter passer being the cause of swine fever is not denied, the view is expressed that the evidence for this being the case is far from compelling. It should be specially pointed out that while in the establishment of the causal relationship of the filtrate, *B. suispestifer* has been considered, no attention has been paid to *B. suissepticus* in this connection.

RECOMMENDATIONS.

Much could possibly be done, in the light of the results obtained in this investigation, to reduce loss from swine fever by—

- (1) Attending to the diarrhoea in sucklings.
- (2) Killing at once, on the first evidence of the condition being present, all “wasters” and “piners” of any degree whatever.
- (3) Attending more closely to the general health of the pigs. Thus, to mention one circumstance, they should be protected from sudden exposure, especially to great cold. For this reason pigs should be kept in pens in large airy houses, entirely under cover during the winter season.

The present paper deals with observations made subsequent

to the publication of the paper just mentioned. The cases referred to in it were derived from four separate outbreaks,—one in Piggery I., two in Piggery II., and one in Piggery III.

Of the pigs mentioned, pig 1 came from Piggery I.; pigs 2, 3, and 4 from Piggery II. (first outbreak); pig 5 from Piggery III.; and pigs 6-85 and sows 1-6 from Piggery II. (second out-break).

Without further premise, therefore, a brief history will now be given of each case, in relation to the clinical symptoms exhibited, so far as these were available, and to the pathological and bacteriological results. Prefixed to each of the piggery groups will be given a short description of the piggery surroundings and the circumstances of the outbreak, so far as known.

PROTOCOLS OF EPIDEMICS.

PIGGERY I.

The epidemic in this case occurred in February, during a time of very severe weather with rain, snow, and frost. The piggery consisted in part of a good covered-in house with separate stalls for the pigs, all under cover, and in part of a wooden lean-to house. This latter had an earthen floor; its lean-to roof was fitted on to the sloping roof of another house in such a manner that all the rain, &c., which collected on this sloping roof ran directly into the pig-house. The wooden walls of the house, in addition, were in bad repair, and had considerable holes in them. The result of such conditions was that the pigs were living all the time in a house with about two inches of water on the floor; there was no dry straw in the place, and what straw was there was more or less incorporated in the mud of the floor, while the rain and snow kept continually trickling down on the occupants. When the place was visited there were no cases in the first-described house. In the second house there were about twelve pigs, just lately weaned. They were covered with mud. Three of them were lying dead and half buried among the water, mud, and straw which formed the floor;

four others were very ill, though able to stand; while the others were obviously ailing. Owing to the mud-stained condition of the animals it was impossible to tell if diarrhoea or a rash was present. The pigs were obviously fevered, as they drank greedily the water from the pools on the floor. Several of them were standing with their hind legs crossed, heads drooping, eyes nearly closed, and chests heaving.

One specimen was obtained from this outbreak—viz., pig 1.

FIG 1.

Small pig; lately weaned; showed during life all the symptoms just described, being one of the group designated as being very ill. It was killed by a blow on the head with a hammer.

Post-mortem.¹—No petechiæ or skin rash was observed: there was a fair amount of subcutaneous fat. Lungs: right middle lobe consolidated, deep red in colour and adherent to the adjacent lobes. Anterior lobe—patches of red consolidation, with remainder of lobe showing acute congestion. Posterior lobe showed acute congestion. Left anterior lobe completely consolidated, grey in colour, with some abscesses and pleurisy. Posterior lobe—acute congestion and marked hæmorrhages. Trachea filled with frothy mucus. Heart apparently healthy. Lymphatic glands all through the body greatly enlarged and deeply red in colour. Abdomen: duodenum inflamed. Small intestine very markedly inflamed and congested, with numerous hæmorrhages. Large intestine inflamed, but no fibrinous exudate or ulcers present. Liver, spleen, gall-bladder, pancreas, kidney, stomach, apparently healthy.

Cultures were made from various organs with the following results: Liver, spleen, gall-bladder, right kidney—no growth. Heart blood (one cubic centimetre inoculated with a pipette)²: Culture tube 1, pure extensive culture of small dewdrop colonies; Tube 2, do. do. Lung: right middle lobe—Tube 1, pure culture of 100 (*circa*) small dewdrop colonies; Tube 2, pure culture of uniform growth small dewdrop colonies; Tube 3, pure culture of 200 (*circa*) small dewdrop colonies. Left anterior lobe—Tube 1, pure culture of 50 (*circa*) small dewdrop colonies; Tube 2, pure culture of 20 small dewdrop colonies; Tube 3, pure culture of uniform growth small dewdrop colonies.

¹ With regard to the post-mortem examinations done in this investigation, it should be noted that, almost without exception, they were performed *immediately* on the death of the animal.

² The technique of the heart-blood culture, in this and in all the cases where it was done, was as follows: The pericardial sac was opened into, the surface of the heart seared, and a sterile glass pipette was plunged through the seared portion of the heart wall, and blood was then sucked up into the pipette by means of a rubber teat. The rubber teat was separated from the cavity of the pipette by means of a cotton-wool plug. About one cubic centimetre of the blood so obtained was inoculated on the surface of an agar tube.

Trachea—Tube 1, pure culture of uniform growth small dewdrop colonies; Tube 2, do. do. Lymphatic glands: bronchial glands—No. 1 gave 4 colonies of the small colony just mentioned in pure condition; No. 2 gave 40 colonies, do. do. Glands at apex of lung—No. 1 gave no growth; No. 2 gave no growth. Cervical glands—No. 1 gave 100 colonies staphylococcus albus + 12 of small colonies just described; No. 2 gave pure extensive culture of staphylococcus albus. Long mesenteric gland gave 1 colony staphylococcus albus + 4 of small colonies just described. Iliac glands—No. 1 gave 6 staphylococcus albus + 14 small colonies; No. 2 gave 8 staphylococcus albus + 4 small colonies. Duodenum (M'Conkey lactose agar plate) gave about equal numbers of a lactose and a non-lactose fermenter. Small intestine gave a lactose and a non-lactose fermenter, but lactose fermenter was in excess.

The reactions of several of the organisms obtained from this case are given in Table I. (p. 6).

The points to be noted in connection with the reactions of the organisms on Table I. are as follows: (1) All the organisms, with the exception of 10, 11, 12, and 13, were gram-negative, pleomorphic, polar-staining cocco-bacilli, which had the common characteristics that they grew on slope agar in small dewdrop-like colonies, and that they rapidly died out on agar, especially if acid (+6 to +10), unless they were subcultured very frequently. The growth of these organisms on agar tended to be different at different times. (2) All these organisms (9, 10, 11, 12, and 13 excepted) did not grow on gelatine at ordinary room temperature. (3) All these organisms (10, 11, and 12 excepted) tended to produce acid in litmus milk, though this took place after a prolonged interval in some cases. (4) Excepting, again, 10, 11, 12, and 13, most of these organisms tended to produce Indol in peptone water. Organisms 6, 7, and 8 did not do this, but their growth was weak in other media. Organism No. 19, however, did not produce it either. Its growth on the other media was weak, though stronger than the three just mentioned. (5) Excepting, again, 10, 11, 12, and 13, there was a tendency for these organisms to produce acid in saccharose. Organisms 6, 7, and 8 did not do this, but their growth in the other media was weak. Organisms 4 and 9 of the more virile strains, however, did not produce this change. (6) There is the further resemblance that all the organisms (with the exception of 9, 10, 11, 12, and 13)

TABLE I.—TABLE OF THE REACTIONS OF THE ORGANISMS FOUND IN FIG 1.

(Heart-blood cultures 1 and 2 were plated out, found to be pure, and individual colonies picked off. Reactions of these colonies given below.)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
	Small colony, trachea.	Small colony, lung, anterior lobe.	Iliac gland, culture 1.	Bronchial gland, culture 1.	Bronchial gland, culture 2.	Cervical gland.	Iliac gland.	Long mesenteric gland.	Pale colony, from intestine.	Pale colony, duodenum 1.	Pale colony, duodenum 2.	Pale colony, duodenum 3.	Pale colony, duodenum 4.	Original culture from heart blood (No. 1).	Original culture from heart blood (No. 2).	Heart blood (No. 1), single colony, A.	Heart blood (No. 1), single colony, B.	Heart blood (No. 1), single colony, C.	Heart blood (No. 1), single colony, D.	Heart blood (No. 2), single colony, A.	Heart blood (No. 2), single colony, B.
Litmus milk . . .	O	A	O	O	A	O	O	O	A	A?	O	A	A	A	A	A	A	O	O	O	O
Inuline . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Glucose . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Dulcitate . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Lactose, neutral red	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Salicine . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Maltose . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Adonite . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Lactose . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Raffinose . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Mannite . . .	A	-	A?	A	A	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Saccharose . . .	A	A	A	A	A	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Galactose . . .	A	A	A	A	A	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Dextrine . . .	A	A?	A?	A?	A	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Sorbit . . .	A	A	O	A	A	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Nitrate water (nitrites)	I	I	I	I	I	O	O	O	I	O	O	O	O	I	-	I	I	I	I	I	I
Peptone water . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Gelatine . . .	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
Litmus milk (in eleven days)	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
	Trace of acid in all of them in eleven days								Acid	Deeply alkaline			Acid	All slightly acid							

A = acid production.
A? = doubtful production.
AG = acid and gas production.

Ac = acid and clot formation.
I = indol formation.
+ = slight nitrite formation or slight

++ = marked nitrite formation or marked growth (no liquefaction).
O = no change or no growth.

were obtained in practically pure massive culture from various situations in *one* carcase of a pig.

The *differences* in the reactions on the various sugars, &c., of these organisms, exclusive of 10, 11, 12, and 13, are, however, very marked. The table has only to be glanced over for this to be seen. Especial attention, however, should be directed to the case of organisms 14, 15, 16, 17, 18, 19, 20, 21. In 14 and 15 the reactions are given of the cultures obtained *direct* from the heart blood of the animal. It will be noticed that the reactions *differ in these two cases* from one another, and that, further, *gas as well as acid* is formed in certain of the media. These two cultures were plated on agar and *found to be pure*. Subcultures were made on ordinary agar *from single colonies*, and the reactions of these colonies are given in 16, 17, 18, 19, 20, 21.

It will be noticed that the reactions of these latter cultures (a) differ from *one another* markedly, (b) differ markedly from the *original cultures* 14 and 15, from which they were derived. In this latter connection it should be noted that *no gas* is formed in any of the media, while in No. 20 the litmus milk is actually clotted—an occurrence which did not take place in the original culture 15.

Reference should now be made to the organisms obtained from the intestine and duodenum in this case. The organisms dealt with in this table from these sources were all gram-negative non-lactose fermenters on M'Conkey lactose plates. The organisms concerned here are Nos. 9, 10, 11, 12, and 13. With regard to organism 9, it will be seen that it has a marked similarity in many points to the group just described, and it should be noted in reference to later developments that its source is the intestine. The resemblances and differences from organisms 10, 11, 12, and 13, also obtained from the intestinal tract, should also be observed. With regard to Nos. 10, 11, 12, and 13, superficially they resemble the so-called classical *B. suispestifer*. Attention should, however, be drawn to the differences between 10 and 11 on the one hand and 12 and 13 on the other. Thus No. 12 produces acid in salicine, and acid only or acid and a small amount of gas in the sugars, &c., in which 10 and 11 produce acid and a large quantity of gas. No. 13 is very unlike No. 12, and

much more unlike 10 and 11. Thus it produces, after a long interval, acid in litmus milk, whereas the other three produce a marked alkaline reaction. Again, it differs from the other three in producing no change in dextrine, and from Nos. 10 and 11 by producing no change in dulcite or sorbite.

It should be further noted that these *suipestifer*-like organisms were not obtained elsewhere in this case than in the duodenum.

PIGGERY II.

This piggery had been free from swine fever for a year. A year previously, however, the disease had broken out, and all the pigs were killed. After the premises had been disinfected a new lot was brought in. No striking disease had been observed in them till about the beginning of March 1916, when a big brood sow became very ill. Her young, which were sucking her, were also affected.

The piggery in this case is an excellent building, taking it all over. Several of the pens, however, communicate with open courts which are not roofed over.

Two points should be noted in connection with this outbreak:—

1st. There was a considerable amount of diarrhoea among the small pigs previous to and subsequent to the sow just mentioned being noticed ill.

2nd. The outbreak took place in the end of February and beginning of March, when the weather was very snowy, frosty, and boisterous.

FIG 2.

This pig, aged about three to four months, was obtained from Piggery II. The animal was very emaciated, was dull, and refused its food. It was killed by a blow on the head on 7th March.

Post-mortem.—No skin rash; very little subcutaneous fat. Lungs apparently healthy. Heart apparently healthy. Lymphatic glands enlarged and congested throughout the whole body. Abdomen: small intestines show little, if any, inflammation. Large intestine—cæcum studded all over with small chronic ulcers. Spleen—minute subcapsular hæmorrhages. Liver and kidneys—healthy, apparently.

Cultures.—Duodenum (on M'Conkey lactose agar): no non-lactose fermenters present; large number lactose fermenters. Lymphatic

glands: gland at apex of lung—culture = single colony small dewdrop. Iliac gland—culture = one staphylococcus, two small dewdrops. Popliteal gland—culture = two staphylococci, four small dewdrops. Cervical gland—culture = two small dewdrops. No cultures were made from heart blood or lung.

The apical gland culture and the iliac gland culture were tested on the various media of Table I. They produced no change in any of them, except a trace of nitrites in nitrate water and a trace of acid in litmus milk.

Morphologically all the small colonies from apical, iliac, popliteal, and cervical glands were alike in that they were gram-negative, pleomorphic, bipolar, cocco-bacilli. Further, they all died out rapidly on agar media, unless they were subcultured at very short intervals.

Organisms resembling the classical *B. suispestifer* were not found in this case.

FIG 3.

This pig was about the same age as pig 2, and came from the same piggery. While it did not appear to be very ill during life, it yet had diarrhoea. It was killed by a blow on the head on 7th March.

Post-mortem.—Nothing abnormal seen except enlargement of the lymphatic glands all over the body and acute inflammation of the small intestine.

Cultures.—Small intestine (on M'Conkey lactose agar): Plate I., 30 colonies, all lactose fermenters; Plate II., 12 do. do. It was not noted if there were differences in size of the lactose fermenting colonies. There were no classical *B. suispestifer* present. No cultures were made from heart blood, lungs, or lymphatic glands. No cultures made from cæcum.

FIG 4.

This pig was from same piggery as pigs 2 and 3, and was about ten weeks old. It died during the night of March 7. It had been noticed to be very ill on the previous day. One special feature present was the very marked paralysis of the hind quarters.

Post-mortem (about five hours after death).—Pig very emaciated; no skin eruptions present. Lungs: Right lung—extensive fibrinous pleurisy and consolidation of whole right lung; left lung—acute congestion. Heart—extensive fibrinous pericarditis. Lymphatic glands enlarged and congested all through body. Abdomen—a very great effusion of clear fluid with flakes in it, into the peritoneum, and a localised peritonitis over an ulcer in the cæcum. Large number of ulcers in cæcum and large intestine. Duodenum, and especially small intestine, very inflamed.

Cultures.—Lungs: Posterior lobe, right side.—Culture 1, pure extensive culture of an organism which showed dewdrop colonies in the first twenty-four hours, but during the next twenty-four hours these colonies enlarged remarkably and became dull and opaque. Cultures 2, 3, and 4 showed the same appearances. Heart blood: culture 1, pure growth of a coliform organism; culture 2, do. do. Lymphatic glands: gland 1=no growth; gland 2=no growth. Duodenum (M'Conkey, lactose agar)—equal numbers of lactose and non-lactose fermenters. Small intestine—equal numbers of lactose and non-lactose fermenters.

TABLE II.—REACTIONS OF ORGANISMS FROM FIG 4.

	1	2	3	4	5	6	7
	Organism from lung.	Heart blood, single colony.	Heart blood, single colony.	Duodenum, single colony.	Duodenum, single colony.	Small intestine, single colony.	Small intestine, single colony.
Litmus milk .	Alkaline	Acid	Acid	Acid	Acid	Acid	Acid
Inuline . .	O	O	O	O	O	O	O
Glucose . .	O	AG	AG	AG	AG	AG	AG
Dulcite . .	O	AG	AG	AG	AG	AG	AG
Lactose, neutral	O	O	O	O	O	O	O
red							
Salicine . .	O	O	O	O	O	O	O
Maltose . .	O	AG	AG	AG	AG	AG	AG
Adonite . .	O	O	O	O	O	O	O
Lactose . .	O	O	O	O	O	O	O
Raffinose . .	O	O	O	O	O	O	O
Mannite . .	O	AG	AG	AG	AG	AG	AG
Saccharose . .	O	O	O	O	O	O	O
Galactose . .	O	AG	AG	AG	AG	AG	AG
Dextrine . .	O	AG	AG	AG	O	O	O
Sorbite . .	O	AG	AG	AG	AG	AG	AG
Peptone (Indol) .	O	O	O	O	O	O	O
Nitrate water for nitrites	++	++	++	++	++	++	++
Gelatine . .	+++	+++	+++	+++	+++	+++	+++
Litmus milk (20 days)	deep blue	deeply Alkaline	deeply Alkaline	deeply Alkaline	deeply Alkaline	deeply Alkaline	deeply Alkaline
Potato . .	Alkaline vigorous copper-coloured	snail track	snail track	snail track	snail track	snail track	snail track

All gram negative cocco-bacilli.

AG=acid and gas formation. +++=very good growth; no liquefaction.
 ++=marked production of. O=no change or no production of.

Symbols as before.

The points to be noted in connection with the reactions of organisms obtained from pig 4 are—

- (1) Organism number 1, obtained in pure massive culture from the lungs, is the Distemper organism.¹
- (2) Organisms numbers 2, 3, 4, 5, 6, and 7, obtained from the heart-blood, duodenum and small intestine of this case, correspond to classical *B. suispestifer*. It is to be noted, further, that there is less variation among them in their reactions than in the case of organisms 10, 11, 12, and 13 of pig 1, Table I.

The following further points have to be noted in connection with this pig:—

- (1) The lumbar region of the cord of this pig shows very marked chromatolysis of the cells of the anterior horn, and also small celled infiltration of the walls of the blood-vessels (see Fig. 20).
- (2) While the lymphatic glands in this pig all over the body showed enlargement and congestion, cultures from them gave no growth. This is of interest from two points of view: firstly, because no *B. suispestifer*-like organism grew; and secondly, *B. suissepticus* may have been present in the glands and yet not have appeared in culture. In this latter case it should be noted that the glands in all the cases observed later, though very enlarged and congested, really contain very few organisms. (Compare in this connection the gland cultures of pig 2.) The pathological change in the gland, moreover, as will be seen later, is of the nature of a hæmorrhage into them. It is not inflammatory in character. Also in this connection it should be borne in mind that *B. suissepticus* is very difficult indeed at times to grow on agar. Thus an apparently viable heavy growth of twenty-four hours' incubation very frequently gives no growth when subcultured on agar immediately.

Vide M'Gowan. Proceedings of Path. Soc. Great Britain and Ireland. Jan. 1910.

„ Journal Path. and Bact. 1911. Vol. xv.

„ Vet. Journal. London, 1912. lxviii. 7.

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PIGGERY III.

FIG 5.

The large intestine and the lung from a pig in an epidemic in this piggery were received. Nothing is known about the conditions in this piggery, nor is the history of the epidemic known, except in so far that swine fever had existed in this piggery for at least three months, and that the pig had received an inoculation of anti-swine fever serum.

Pathological changes: lungs—a condition of grey consolidation existed; colon—large number of small healing ulcers.

Cultures.—Several cultures were made from the lung. In all of them two types of colony grew in abundance: (1) small dewdrop-like colonies which remained small in size after forty-eight hours; (2) small dewdrop colonies at the end of twenty-four hours, which, however, increased very rapidly in size and opacity during the next twenty-four hours.

TABLE III.—FIG 5. REACTIONS OF ORGANISMS.

	Organism No. 1.	Organism No. 2.		Organism No. 1.	Organism No. 2.
Litmus milk . . .	Acid	Alkaline	Galactose . . .	O	O
Inuline . . .	O	O	Dextrine . . .	O	O
Glucose . . .	O	O	Sorbite . . .	O	O
Dulcitate . . .	O	O	Peptone (Indol) . . .	O	O
Lactose, neutral red	O	O	Nitrate water (nitrites)	+	+
Salicine . . .	O	O	Gelatine . . .	O	++
Maltose . . .	O	O	Litmus milk (20 days)	slight Acid	deeply Alkaline
Adonite . . .	O	O	Potato . . .	O	vigorous copper- coloured
Lactose . . .	O	O			
Raffinose . . .	Acid	O			
Mannite . . .	O	O			
Saccharose . . .	O	O			

O = not present or no change. + = present. ++ = very good growth.

Symbols as before.

The points to note in connection with these reactions are—

- (1) Organism No. 1 resembles the small dewdrop cultures of Tables I. and II.; it also resembled them morphologically.
- (2) Organism No. 2 is again the distemper organism, obtained this time from a case from a quite different piggery.

PIGGERY II. (second outbreak).

The first outbreak, which has been dealt with above, occurred in the beginning of March. The pigs were all killed out at this time, and the piggery thoroughly cleaned and disinfected during the next month and a half.

From about April 17 to the beginning of May new batches of pigs began to be brought in. These new pigs came from four sources—A, B, C, and D. No swine fever was known to exist in any of these sources.

The pigs were of various ages, as follows:—

Under two months — 37 pigs, hereafter designated group β .

Under four months but above two—21 pigs, hereafter designated group γ .

Under six months but above four, including six gilts in pig—15 pigs, hereafter designated group δ .

Total number of pigs brought in = 73.

Of these groups the following numbers of each were obtained from the various sources:—

From source A,	13	belonging to group γ .
From source B,	9	„ δ .
From source C,	37	„ β .
From source D, {	6	„ δ .
	8	„ γ .

VACCINATION.

The owner of the pigs was very anxious that something in the way of vaccination should be tried to see if these pigs could not be prevented from taking the disease. Having, therefore, in view the fact that I had obtained the distemper bacillus in the case of the two pigs just mentioned, I thought that, having regard to the results claimed for a vaccine made from the distemper bacillus in distemper of dogs, a similar vaccine would be worth trying here. On the other hand, I thought it would not be well to leave out of consideration in the preparation of the vaccine the two organisms *Bacillus suisepiticus* and *B. suispestifer*.

The owner readily agreed to my proposals to use this

vaccine, and a vaccine composed of equal numbers of twenty-four-hours' cultures of the bacillus of distemper from the pig (two strains), of *B. suis* (six strains), *B. suis* (eight strains), was made up. The cultures were emulsified in saline; this emulsion was put into a sterile tube; to about 20-30 cubic centimetres of this emulsion about 3 cc.'s chloroform was added. The tube was then sealed off, and the emulsion shaken up vigorously with the chloroform at intervals for two days.

After this treatment, the emulsion was tested two or three times for sterility, and on each occasion it was found to be sterile.

This emulsion was then used for inoculation of the pigs in the following doses:—

Pigs of group β received $\frac{1}{16}$ th of a twenty-four-hours' agar culture of each organism for the first dose.

Pigs of the other groups (γ and δ) received double the dose of the pigs in group β .

Where a second injection was performed it was done a week after the first and consisted of a double dose.

Method of vaccination.—A strong solution of iodine in alcohol was painted on the loose skin behind and below the ear, or on the skin in the inside of the thigh, and the injection made in these situations.

Immediate effects of vaccination.—Generally none was noticed; there was no local swelling or redness, and as a general rule the pigs were taking their food quite well next day. In one group the pigs were a little off their food next day, but were back to it again on the next.

The pigs were inoculated between April 18 and May 17, and in all 50 were inoculated, made up as follows:—

Group β , 22 out of 37, leaving 15 uninoculated.

Group γ , 13 „ 21, „ 8 „

Group δ , 15 „ 15, „ 0 „

— — —
Total, 50 „ 73, „ 23 „

The pigs remained fairly well till about May 6, when pig 6, a pig of group γ and from source A, became ill and refused its food. Meantime it should be noted that the weather was very good.

FIG 6.

A pig of group γ and from source A. Vaccinated April 18, and again May 3. Three days after last inoculation, on May 6, it became ill and refused its food. May 9. Temperature is 107° F.; constipated; a bluish-red raised rash, which disappears on pressure, situated all over under surface of body and in groins and axillæ. May 10. Rash still on abdomen; temperature=107° F. Animal remained in much the same condition till the night of May 14-15, when it died.

Post-mortem (a few hours after death).—The rash described above had disappeared from the abdomen, but there was a marked petechial rash in the axillæ and groins. Lungs: extensive effusion of a clearish fluid, with a coagulum floating in it, into right pleural cavity. Right lung deep red and consolidated throughout. Left lung—congestion (but no chronic pneumonia present in the fringes of this lung). Heart—excess of yellow pericardial fluid; petechial hæmorrhages on surface of auricles. Lymphatic glands enlarged and reddened all through body. Abdomen: intestines—marked hæmorrhages and inflammation all down intestine from stomach to cæcum and colon. Large submucosal hæmorrhages in cæcum and colon, but no ulcers present. Other abdominal organs apparently healthy, except that spleen is slightly enlarged. Tongue—marked ulceration, with white sloughs at the root of the tongue.

Cultures.—Lung (right): culture No. 1, pure extensive culture of small grey dewdrops; culture No. 2, do. do.; culture No. 3, do. do. Trachea: culture No. 1, practically pure extensive culture of small dewdrop colony; culture No. 2, do. do. Lymphatic gland—abdomen—culture gave no apparent growth.

TABLE IV.—REACTIONS OF ORGANISMS FROM FIG 6.

	LUNG.			LUNG.	
	Culture No. 1.	Culture No. 2.		Culture No. 1.	Culture No. 2.
Litmus milk . . .	O	O	Mannite	O	O
Inuline	O	O	Saccharose . . .	O	A
Glucose	O	O	Galactose	O	O
Dulcite	O	O	Dextrine	O	O
Lactose, neutral red .	O	O	Sorbite	O	O
Salicine	O	O	Peptone (Indol) .	O	+
Maltose	O	O	Nitrate water (nitrites)	+	+
Adonite	O	O	Gelatine	O	O
Lactose	O	O	Litmus milk (20	A?	A
Raffinose	O	O	days).		
	1	2		1	2

Gram-negative pleomorphic polar-staining cocco-bacilli, rapidly dying out on agar if not subcultured.

Symbols as before.

The special points in connection with this case are the following: that the disease began three days after the second inoculation; it was the first case in this outbreak; it was a very acute case; it showed skin eruptions and ulceration at the root of the tongue; the distemper bacillus was not found; and there was no evidence of a chronic pneumonic condition in the fringes of the left lung.

FIG 7.

Pig from source A and of group γ ; also a pen-mate of pig 6. It was vaccinated April 18 and again on May 3. First noticed ill on May 15, nine days after pig 6 was first seen to be ill. On this date it was dull and off its food. No rash was present. Its temperature was 105° F. May 17, temperature was 106.1° F. It was very shaky on its legs; its nose was twisted to one side; there was no rash and no diarrhoea; its nose and eyes were very dirty from a discharge. After it had been handled a little it fell down from weakness, but after resting a little it got up again. It was killed on May 17.

Post-mortem.—No skin rash. Lungs: right lung—œdema and congestion (no chronic pneumonia present); left lung—no change (no chronic pneumonia present). Heart apparently healthy. Lymphatic glands enlarged and congested. Abdomen—congestion of small intestine.

Cultures.—Nine cultures were made from the lung, but no growth was obtained from them. No cultures were made from any other situation.

Special points in this case are—that this pig was affected with an acute illness; the illness began twelve days after last inoculation and nine days after pig 6 was noticed to be ill (question of infection from pig 6); the absence of chronic pneumonic lesions in the fringes of the lungs; the very great weakness exhibited, together with the twisting of the nose to one side (? paralytic condition).

FIG 8 and 8¹.

Pigs from source A and of group γ ; pen-mates of pigs 6 and 7. Vaccinated April 18 and again May 3. First noticed ill about May 18, being dull and not taking their food. This condition continued for two or three days, when it was noted on May 22 that they were very much better, and again on June 1 that they appeared all right again. After this they continued in good health, and were killed as fat on July 8.

Post-mortem.—The carcasses were healthy, nothing abnormal being found either in lungs or intestine.

FIG 9.

Pig from source C and of group β . It had been vaccinated on May 10 and May 17. It was noticed to be ill. On May 21 it refused its food; its eyes were bleary and watery; there was a discharge from the nose. It was killed by pithing on May 22.

Post-mortem.—Purulent discharge from eyes and nose (see Fig. 1). All over the body, but especially beneath the abdomen, there were raised large dark coloured spots, about the size of a shilling piece, covered with scales (see Fig. 2). Lungs: Right—lower margins, middle lobe, and postcardial lobe, solid, pale grey, and of a consistency and appearance like pancreas.¹ On section, abundant mucopus exuded from the bronchi; Left—lower margins in same pancreas-like condition. Heart—apparently healthy. Lymphatic glands enlarged and reddened all over, especially in the thorax. Abdomen—very considerable amount of clear fluid with flakes of coagulum in it in the peritoneal cavity; one or two ulcers in the cæcum. No hæmorrhages seen anywhere in body. Other organs in abdomen appear healthy.

Cultures.—Lung: twelve cultures in all made from different parts of the lung; with the exception of a few colonies of a staphylococcus in some of the tubes, practically all the tubes gave pure extensive cultures of small dewdrop colonies.

Special points in this case are—that this was the first among group β to be obviously ill; the presence of discharge from the nose and eyes as a symptom; the distemper bacillus, though looked for in cultures from the trachea and nose, was not found; the presence of the rash in the form of large raised dark spots; active symptoms of illness beginning to show four days after the second vaccination; the age of the lesions in the lungs with reference to the beginning of the disease in this case.

FIG 10.

Pig from source C and of group β . It had been vaccinated on May 10 and 17. This pig was very small and ill-thriven. It was first noticed acutely ill on May 21, when it was seen to be dull and not eating. There was also a discharge from the eyes and nose. Killed by pithing on May 24.

Post-mortem.—Pig very emaciated; discharge from eyes and nose; no rash. Lungs: Right—lower borders, middle lobe, and postcardial lobe “pancreas-like”; Left—lower borders “pancreas-like.” Heart

¹ Hereafter this appearance will be noted as “Pancreas-like.”

apparently healthy. Lymphatic glands, especially in thorax, enlarged and reddened. Abdomen—nothing abnormal seen.

Cultures.—Twenty-eight cultures in all were made from various parts of the lung. With the exception of a few colonies of staphylococci, all the cultures gave pure extensive growths of a small dewdrop colony. The distemper bacillus was not present.

The special points in this case are—

- (1) Much the same condition present here as in pig 9, with the exception of the spots.
- (2) In this case, in connection with the possible origin of the disease, it should be noted that—
 - (a) The pig was obviously a pinner; it was undersized and emaciated.
 - (b) Active symptoms came on about two days after the second dose of the vaccine was administered.
 - (c) The lesions found post-mortem were of a subacute type, and must have been present prior to the arrival of the pig at this piggery on or about May 9.

FIG 11.

Pig from source C and of group β . Had been vaccinated on May 10 and 17. It was a pen-mate of pigs 9 and 10. On June 2 it was noted that it had been acutely ill for the last few days. There was purulent discharge from the nose, and the eyelids were gummed together in the morning. It had been taking no food lately. The animal was highly fevered; cough was present; there was a fine purplish coloured rash, especially on the abdomen, which rash disappeared on pressure. It was killed by pithing on June 2.

Post-mortem.—Lungs: right middle lobe, postcardial lobe, and part near the root of anterior lobe were reddened, consolidated, but tough. There was abundant purulent exudate from the bronchi. Heart apparently healthy. Lymphatic glands throughout the body were enlarged and reddened. Abdomen: peritoneal fluid very greatly increased in quantity; several pints present of a clearish fluid containing coagula. The peritoneum over cæcum, ileocaecal valve, and colon was very oedematous and inflamed. In the cæcum and colon there were numerous large old-standing black button ulcers. The other abdominal organs were apparently healthy.

Cultures.—Twelve cultures were made from the lung in this case. In most of these nothing grew, but in some of them there were a few colonies of a staphylococcus. No cultures were made from heart blood, lymphatic glands, or intestine.

Special points to note (in reference to the possible origin of the disease) are—

- (1) The retrogressive nature of the lesions in the lungs as evidenced by—
 - (a) The absence of growth in the cultures.
 - (b) The tough nature of the lung tissue.
- (2) The old-standing nature of the lesions in the intestine.

FIG 12.

Same history as in pig 11. Killed June 2 by pithing.

Post-mortem.—Lungs: right middle lobe, postcardial lobe, and lower margin of anterior lobe were pale, slightly consolidated, but tough and crepitant. The condition suggested a pneumonic condition nearly resolved. Heart apparently healthy. Lymphatic glands enlarged and reddened. Abdomen: peritoneum contained a very great excess of clear fluid, with coagula present. There was localised peritonitis over several large button ulcers in the cæcum.

Cultures.—Fourteen cultures were made from various parts of the lung. In the most of them nothing grew, but in a few there were a few colonies of a staphylococcus.

A special point in this case (in reference to possible origin of the disease) is the resolving condition in the lung and the chronic old-standing condition in the intestines.

FIG 13.

Pig from source C and of group β , was a pen-mate of pigs 9, 10, 11, and 12 until June 2, when it was removed, along with pig 14, to a separate pen. On June 12 it was noted that it was very dull and not taking its food; its eyelids were gummed together in the morning, and it was becoming very thin. June 15. Very weak and ill; standing with hind legs crossed; very emaciated; taking no food. Killed on June 16 by pithing.

Post-mortem.—Very emaciated. Lungs—effusion into pleural cavities on both sides. Right—middle lobe, lower border anterior lobe, and postcardial lobe “pancreas-like”; Left—lower and anterior borders of left lung “pancreas-like.” Heart apparently healthy—no pericarditis. Lymphatic glands enlarged and reddened all through the body. Abdomen—great effusion of fluid, with coagula, into peritoneal cavity; very large black button ulcers at ileocæcal valve; hæmorrhagic spots dotted all through the substance of the liver.

Cultures.—None made.

FIG 14.

Same history as in pig 13. On June 10 it was noted that there were numerous subcutaneous hæmorrhages. Its eyelids were gummed together, it was not taking food, and it was becoming very thin. On June 15 it was very ill, standing with its hind legs crossed. On June 16 it was nearly dead, it was cold to the touch, and its ears were quite blue: it was killed by pithing.

Post-mortem.—Very emaciated; there were a few subcutaneous hæmorrhages about the heels. Lungs—condition as in pig 13. Heart straw-coloured effusion into pericardial sac, and definite early pericarditis all over the surface of the heart. Lymphatic glands enlarged and congested all over body. Abdomen—great effusion of clear fluid, with coagula, into peritoneal sac, and slight peritonitis over some very large black button ulcers in cæcum and at ileocæcal valve. Minute pin-point hæmorrhages all over the small intestine.

Cultures.—No cultures made.

Special points in pigs 13 and 14 are—the presence of chronic old-standing lesions with more acute changes super-added; the presence of pericarditis in pig 14. The fact should further be noted that all the pigs of group β which were acutely ill and on which a post-mortem has been done up to the present time were derived from one pen, a point to be emphasised in connection with the spread of the disease.

FIG 15.

This was a boar from source A and of group γ . It was vaccinated on May 3 and again on May 10. It was first noticed ill on June 12, and remained more or less ill till June 21. The symptoms exhibited were dulness, want of appetite, and diarrhoea. From June 21 it remained fairly well till July 27, when it again became ill. It was dull, would not eat its food, remained inside its house and would not come out into the court. In July 28 it was further noticed that there was a large hæmorrhage on the scrotum and one on the back of the ear. From July 28 till August 9 the animal remained in much the same condition. No diarrhoea was observed at any time during this attack. On August 9 it was noticed to be definitely lame in a hind leg, and this lameness persisted and meantime affected the other hind leg till August 25, when the animal was killed. At the same time more or less of dulness and loss of appetite were also present.

Post-mortem.—The only lesions found post-mortem were a very small piece of resolving grey consolidation in the right middle lobe and in the postcardial lobe. The lymphatic glands at the root of the lung were enlarged and reddened. The intestines were perfectly healthy. There were no lesions in the hind limbs to account for the lameness.

Cultures.—No cultures were made.

Special points in this case are—the presence of diarrhœa; the presence of lameness in both hind legs; and the practical absence of lesions post-mortem, with the exception of the small patches in the lung.

Pig 16.

Pig from source C and of group β . Had been vaccinated on the 10th of May and again on the 17th. It was first noticed to be dull and refusing food on the 10th of June. It remained in much the same condition till June 19, with the additional symptom that its eyelids were gummed together in the morning. It was killed by pithing on June 19.

Post-mortem.—It was a very small and emaciated pig. Lungs—chronic “pancreas-like” consolidation of the right middle lobe and lower margins of right anterior lobe. Left lung—similar condition in lower borders. Heart apparently healthy. Lymphatic glands enlarged and reddened throughout body. Abdomen—large amount of clear fluid with coagula in peritoneal sac. Localised peritonitis over colon and cæcum. Cæcum and colon lined with a diphtheritic deposit.

Cultures.—Lung: middle lobe right side—Culture No. 1, pure extensive culture of small dewdrops; No. 2, do. do. Left lobe—Culture No. 1., do. do.

TABLE V.—REACTIONS OF ORGANISMS FROM Pig 16.

	LUNG.			LUNG.	
	Right middle lobe.	Left lobe.		Right middle lobe.	Left lobe.
Litmus milk . . .	A ?	O	Raffinose	O	O
Inuline	O	O	Mannite	O	O
Glucose	A	A	Cane sugar	O	O
Dulcite	O	O	Galactose	A	A
Lactose, neutral red .	O	O	Dextrine	O	O
Salicine	O	O	Sorbite	O	A
Maltose	O	O	Peptone (Indol) .	+	+
Adonite	O	O	Nitrate water (nitrites)	+	+
Lactose	O	O	Gelatine	O	O
	1	2		1	2

Symbols as before.

Special points in this case are the discharge from the eyes and nose, and the diphtheritic deposit in cæcum and colon.

FIG 17.

Pig from source C and of group β was unvaccinated. First noticed actively ill on June 10. Symptoms exhibited between 10th June and 19th, when it was killed, were dulness, want of appetite, heaving of sides, and discharge from eyes and nose. Killed by pithing on 19th June.

Post-mortem.—Lungs same condition as in pig 16. Heart apparently healthy. Abdomen apparently healthy, except for congestion of cæcum and a small ulcer on the ileocæcal valve. Lymphatic glands enlarged and reddened, especially in thorax.

Cultures.—Lungs: Culture No. 1, pure extensive culture of small dewdrops; No. 2, do. do.; No. 3, do. do.; No. 4, do. do. No cultures made from other organs.

TABLE VI.—REACTIONS OF ORGANISMS FROM FIG 17.

	Lung culture.		Lung culture.
Litmus milk	O	Mannite	O
Inuline	O	Saccharose	A
Glucose	A	Galactose	O
Dulcite	O	Dextrine	O
Lactose, neutral red	O	Sorbite	A?
Salicine	O	Peptone (Indol)	+
Maltose	O	Nitrate water (nitrites)	+
Adonite	O	Gelatine	O
Lactose	O	Litmus milk (20 days)	A
Raffinose	O		

Symbols as before.

The special point in this case is that this was the only one of the unvaccinated pigs of group β which died.

FIG 18.

Pig from source C and of group β , vaccinated May 10 and 17. General symptoms exhibited from June 12 till July 10, when it was killed, were dulness, want of appetite, and discharge from nose and eyes. On June 27 it developed an abscess at the upper margin of the hoof, which did not heal. Killed on July 10 by pithing.

Post-mortem.—Lungs—"pancreas-like" condition in usual situations. Heart—small hæmorrhages on its surface. Lymphatic glands enlarged and reddened all through body. Abdomen—chronic peritonitis, with a localised abscess over the large intestine and adhesions throughout peritoneum. Petechial hæmorrhages all down small intestine. Ulcers in cæcum and colon. Spleen much enlarged. Kidney—"turkey egg" appearance due to small hæmorrhages.

Cultures.—No cultures made in this case.

FIG 19.

Pig from source C and of group β , vaccinated May 10 and 17. First noticed ill on June 12. Symptoms between June 12 and July 25, when it was killed, were dulness, want of appetite, discharge from eyes and nose. On July 25 the body temperature fell, the animal became quite cold to the touch, and its ears became deep blue. Killed by pithing on July 25.

Post-mortem.—Ulcer with white necrotic surface on tip of tongue (Fig. 12), and near it subepithelial hæmorrhages quite visible to the naked eye. Ulcers at side and root of tongue and subepithelial hæmorrhages at root of tongue and epiglottis. Marked hæmorrhages into tonsils. Lungs—"pancreas-like" change, but in this case deeply reddened, in the usual situations. Acute pleurisy all over surface of the lungs on both sides. Heart—large amount of straw-coloured fluid in pericardial sac. Lymphatic glands enlarged and reddened all over body. Abdomen—large chronic black ulcers along the whole length of cæcum and colon; minute subepithelial hæmorrhages in stomach. Spleen enlarged and soft. Kidneys marked "turkey egg." Bladder—very marked subepithelial hæmorrhages all over surface of bladder (Figs. 4 and 5).

Cultures.—No cultures made in this case.

The special points in this case are the ulcers and hæmorrhages in the mouth, and the marked subepithelial hæmorrhages in the bladder.

FIG 20.

Pig from source B and of group δ , vaccinated. (This pig, along with pigs 21, 22, 23, had never shown a day's illness since coming to the piggery. They were killed because of the fear that they might take the disease. They weighed about twenty stone when killed.) Killed June 15.

Post-mortem.—Entirely healthy.

FIG 21.

Same source and history as in pig 20. Killed June 15.

Post-mortem.—entirely healthy.

FIG 22.

Same source and history as in pigs 20 and 21. Killed June 15.

Post-mortem.—Right middle lobe consolidated and pancreas-like.

The special point in this last case is the condition in the lung, in spite of which the animal showed no sign of disease, thrived and grew fat, although it had a cough.

FIG 23.

Same source and history as in pigs 20, 21, and 22. Killed June 15.

Post-mortem.—Lungs and heart—remains of an old right-sided pleurisy and pericarditis and adherent pericardium, both conditions entirely quiescent now.

The special point in this case is the presence of this old-standing pleurisy and pericarditis, evidence of the animal having been affected with a disease of a similar nature to the one found in the small pigs before the animal came to the piggery. The animal showed no evidence of disease while it was at the piggery, and thrived and grew fat.

FIG 24.

Pig from source A and of group γ ; had not been vaccinated. First noticed actively ill on June 14; was dull and off food. Killed on June 15.

Post-mortem.—Heart and lungs healthy, but cæcum and colon have numerous large black old-standing ulcers with enlarged and reddened lymphatic glands.

The special point in this case is that this pig, in spite of these lesions, was noticed ill only on the 14th. Previous to this it had appeared quite well.

FIG 25.

Pig from source C and of group β , vaccinated May 10 and 17. First noticed actively ill on June 20; previous to this, however, it had not been thriving, as it was very dwarfed in size. On June 20 it became dull, and refused food, and began to breathe heavily. It remained in much the same condition till June 23, when it was killed by pithing.

Post-mortem.—Lungs—old-standing adherent pleurisy on right side; lower margins of lungs on both sides and middle lobe right side are “pancreas-like.” Heart—old-standing adherent pericarditis. Lymphatic glands enlarged and reddened throughout body. Abdomen—very large quantity of clear fluid with yellowish coagula. Marked ulcers in colon, with localised peritonitis over them.

Cultures.—Six cultures were made from the lungs in this case. In them all there grew a pure extensive culture of small dewdrop colonies. There were, however, all gradations in size of the colonies, from pin-point colonies to colonies with exactly the same appearance but of six times the diameter. These colonies are named the small

and the large variety, and their cultural reactions are given in Table VII. Morphologically they had all the same appearance, that of a pleomorphic gram-negative cocco-bacillus.

TABLE VII.

	Small colony lung.	Large colony lung.		Small colony lung.	Large colony lung.
Litmus milk . . .	O	O	Mannite . . .	O	A
Inuline . . .	O	O	Saccharose . . .	A	A
Glucose . . .	A	A	Galactose . . .	A	A
Dulcite . . .	O	O	Dextrine . . .	A	A
Lactose, neutral red.	O	O	Sorbite . . .	A	A
Salicine . . .	O	O	Peptone (Indol) . .	+	? +
Maltose . . .	O	O	Nitrate water (nitrites)	+	+
Adonite . . .	O	O	Gelatine . . .	O	O
Lactose . . .	O	O	Litmus milk (10 days)	A	A
Raffinose . . .	O	O			

Symbols as before.

The special point in this case is the occurrence of large and small colonied varieties of the same organism in the lung of this case.

FIG 26.

Sow from source B and of group δ ; was vaccinated. Had been doing well, but it was noticed to be getting thinner lately and also to possess a loose open-mouthed cough. Killed on June 29.

Post-mortem.—"Pancreas-like" condition in both lungs in the usual situations. Lymphatic glands in thorax enlarged and congested. Abdominal organs all appear healthy. No cultures made in this case.

FIG 27.

Pig from source A and of group γ ; was not inoculated. It was first noticed ill on June 24, owing to its dulness and refusal to take food. On June 27 the additional symptom of lameness in the hind legs appeared. There was no abscess in the feet, and the leg was not painful when handled. Killed June 29.

Post-mortem.—Lungs—right middle lobe, and lower and anterior margins of lungs on both sides for a considerable depth are solid, hard, and friable, and of a deep red colour. The postcardial lobe is in same condition. There is also a pleurisy on the right side. Heart apparently healthy. Lymphatic glands enlarged and reddened all over body. Abdomen: small intestine deeply inflamed; cæcum and large intestine—no ulcers present, but mucous membrane uni-

formly and deeply inflamed and discharging blood on the surface. The faeces in the large intestine are rolled up into hard, angular, tarry-coloured balls, which are sticking to the surface of the mucosa.

Cultures.—Two cultures made from the lung in this case gave pure extensive cultures of small dewdrop colonies.

The special points in this case are the presence of lameness and the special condition found in the large intestine.

FIG 28.

Pig from source A and of group γ ; was not vaccinated. Was a pen-mate of pig 27. First noticed ill on June 24; symptoms were dulness and inappetence. Killed June 29.

Post-mortem.—Lungs—"pancreas-like" condition in usual situations. Enlarged lymphatic glands in thorax, but none in abdomen. Abdomen—all organs apparently healthy. No culture made in this case.

FIG 29.

Pig from source C and of group β ; was vaccinated 10th and 17th May. Had been ill for some time with dulness and inappetence. Killed June 29. Post-mortem appearances as in pig 28.

FIG 30.

Same history as in pig 29, and same post-mortem appearances.

FIG 31.

Same history as in pigs 29 and 30.

Post-mortem (June 29).—Same post-mortem appearances as in pigs 29 and 30, except that there was present here one very large button ulcer in the large intestine. The surface of the intestine surrounding this ulcer was diffusely inflamed for a considerable distance, and here and there on the surface of this inflamed mucosa were little white tags of membrane, adherent by pin-point attachments to the mucosa.

Cultures.—Twelve cultures were made from different parts of the lung. Of these, eight gave pure uncontaminated extensive cultures of a small dewdrop colony, while the other four were slightly contaminated with a few colonies of a staphylococcuseus.

FIG 32.

Pig from source D and of group γ ; was not vaccinated. First noticed actively ill on June 24. Symptoms were dulness and inappetence. Killed on June 29.

Post-mortem.—Very small pig for its age. Lungs—"pancreas-like" condition in usual situations. Lymphatic glands enlarged and reddened in thorax. Abdomen—all organs appear healthy. No cultures made in this case.

FIG 33.

Pig from source C and of group β ; vaccinated 10th and 17th May. Previous to 16th June it had been running at eyes and nose, but was not off its food. On June 16 it became dull and refused food, and remained in this condition till it was killed on June 29.

Post-mortem.—Lungs—"pancreas-like" condition in usual situations. Heart—adherent pericarditis, old-standing and quiescent. Lymphatic glands enlarged and reddened in thorax. Abdomen—organs apparently healthy. No cultures made in this case.

The special point in this last case is the presence of the old-standing pericarditis, which had existed prior to June 16, and yet the animal had not showed any symptoms which one would associate with such a grave condition.

FIG 34.

Pig from source C and of group β . Prior to June 30 this pig had been ailing for a long time, showing dulness, lack of appetite, rashes, and discharge from eyes and nose. On June 30 it was noticed to be very ill—body cold, ears blue, with large raised dark-coloured ulcerating spots all over the body, especially in axillæ and groins. Killed by pithing.

Post-mortem.—Lungs—red acute pneumonic consolidation of right posterior and middle lobes and of lower and anterior borders of right and left lungs. Acute pleurisy on both sides. Heart—pericardial sac distended, with deep red-coloured fluid containing coagula; commencing pericarditis and hæmorrhages all over surface of heart. Lymphatic glands all over body enlarged and reddened. Abdomen—minute subperitoneal hæmorrhages, punctate in character all over surface of abdominal organs, especially the intestines. Punctate submucosal hæmorrhages in stomach. Large intestine—numerous large dark-coloured button ulcers of some standing; also numerous large submucosal hæmorrhages bulging into the lumen of the intestine (see Fig. 8). Spleen—enlarged, dark brown, and soft. Kidney—"turkey egg," with hæmorrhages into the papillæ. Bladder—no hæmorrhages into mucosa.

Cultures.—Gland 1: culture = 1 colony of small dewdrop. Gland 2: culture = 200 colonies of small dewdrop and 15 staphylococci. Lungs—two cultures were made from the left lung and four cultures from right; all these cultures gave a practically pure uniform growth of small

dewdrop colonies, except that in some of them there were a few colonies of staphylococci.

TABLE VIII.—REACTION OF ORGANISMS FROM FIG 34.

	Gland culture.	Lung culture.		Gland culture.	Lung culture.
Litmus milk . . .	O	A	Mannite	O	O
Inuline	O	O	Saccharose	A	A
Glucose	O	O	Galactose	O	A
Dulcitate	O	O	Dextrine	O	O
Lactose, neutral red .	O	O	Sorbite	O	O
Salicine	O	O	Peptone (Indol) .	?	+
Maltose	O	O	Nitrate water (nitrites)	?	+
Adonite	O	O	Gelatine	O	O
Lactose	O	O	Litmus milk (10	A	A
Raffinose	O	O	days).		
	1	2		1	2

Both organisms pleomorphic gram cocco-bacilli.
Symbols as before.

The special point in this case is the presence of large sub-mucous hæmorrhages bulging into the lumen of the large intestine, especially in relation to the question of the genesis of the ulcers.

FIG 35.

Pig from source C and of group β ; vaccinated 10th and 17th May. During May and June it had been affected with discharge from the nose and eyes. It appeared to get better from this for a while; then about the end of June it became dull and refused its food, meantime developing extensive hæmorrhagic rashes of a deep purple colour. For several days before death the ears were intensely livid in colour and cold to the touch. It died during the night of July 3-4.

Post-mortem (July 4, a few hours after death).—Blue and black patches all over the body, some of them as large as an ordinary saucer. When cut into they were found to be hæmorrhages. There was a spot on the moist portion of the nose about the size of a shilling piece, which was livid at the periphery and white and necrotic in the centre (see Figs. 10 and 11). Lungs—lower margins of right lobe, right middle lobe, postcardial lobe, and lower and anterior borders of left lung consolidated, friable, and greyish red; pleurisy on right side. Heart apparently healthy. Lymphatic glands enlarged and reddened throughout whole body. Abdomen—large black ulcers of some standing in cæcum and colon. Other abdominal organs apparently healthy.

Cultures.—Two cultures were made from the lungs, and both gave pure extensive cultures of small dewdrop colony.

The special point in this case is the occurrence of the necrotic patch on the nose, especially in reference to the genesis of the ulcers.

FIG 36.

Had much the same history as pig 35—same source and group, same pen, same rash, and same blueness of ears. Killed July 4.

Post-mortem.—Animal very emaciated and ears very blue. Lungs—same condition as in pig 35; pleurisy present. Heart apparently healthy. Lymphatic glands enlarged and reddened all over body. Abdomen—All organs apparently healthy.

Cultures.—Two cultures were made from the lungs, and these gave practically pure extensive cultures of small dewdrop colony. There were, however, a few contaminating colonies of a staphylococcus albus present.

TABLE IX.—REACTIONS OF ORGANISM FROM FIG 36.

	Lung cul- ture, small dewdrop.		Lung cul- ture, small dewdrop.
Litmus milk	O	Mannite	A
Inuline	O	Saccharose	A
Glucose	O	Galactose	A
Dulcite	O	Dextrine	O
Lactose, neutral red	O	Sorbite	A
Salicine	O	Peptone (Indol)	+
Maltose	O	Nitrate water (nitrites)	+
Adonite	O	Gelatine	O
Lactose	O	Litmus milk (10 days)	A
Raffinose	O		

Symbols as before.

FIG 37.

Same history as pigs 35 and 36. It was the largest and liveliest of the three. About a week ago (from July 4) it had numerous small subcutaneous hæmorrhages all over the body and was very livid in the ears. These had disappeared by July 4, when the animal was killed.

Post-mortem.—Lungs—"pancreas-like" in usual situations. Heart—no pericarditis; apparently healthy. Lymphatic glands all over body enlarged and reddened. Abdomen—a few button ulcers in cæcum and colon. Other abdominal organs apparently healthy.

Cultures.—Two cultures made from lung; both gave pure uncontaminated extensive cultures of small dewdrop.

TABLE X.—REACTIONS OF ORGANISM FROM FIG 37.

	Lung culture, small dewdrop.		Lung culture, small dewdrop.
Litmus milk	O	Mannite	A
Inuline	O	Saccharose	A
Glucose	O	Galactose	A
Dulcitate	O	Dextrine	O
Lactose, neutral red	O	Sorbite	A
Salicine	O	Peptone (Indol)	+
Maltose	O	Nitrate water (nitrites)	+
Adonite	O	Gelatine	O
Lactose	O	Litmus milk (10 days)	A
Raffinose	O		

Symbols as before.

FIG 38.

Pig from source B and of group δ; vaccinated. Never showed any signs of active illness; was dull and not thriving, and had at times a loose open-mouthed cough. Killed July 8 by butcher.

Post-mortem.—Lungs—“pancreas-like” lesions in usual places, but more restricted, and lung somewhat crepitant. Abdomen—large old-standing ulcers in large intestine. No cultures made in this case.

FIG 39.

Pig from source A and of group γ; was not vaccinated. First noticed dull on July 7. Previous to this there had been no active signs of illness. Killed by butcher on July 8.

Post-mortem.—Lungs—“pancreas-like” lesions in usual places. Abdomen—cæcum and colon uniformly inflamed all over, with surface weeping blood, and hard angular tarry lumps of fæces adherent to the mucosa. No ulcers present. No cultures made in this case.

FIG 40.

Pig from source D and of group γ; was not vaccinated. Was first noticed to be ill on June 27. On July 7 it was noticed to be very dull and ill, and its sides were heaving. Killed July 8.

Post-mortem.—Lungs—“pancreas-like” condition in usual situation. Abdomen—large intestines, old-standing ulcers at ileocæcal valves. No cultures made in this case.

FIG 41.

Pig from source C and of group β ; was not inoculated. It was one of four very small "waster"-like pigs obtained from source C. Up to July 1, however, this animal ate its food greedily, but never seemed to grow. Latterly it became dull, apathetic, refused its food, and coughed a good deal. It was killed on July 22, as it was very ill.

Post-mortem.—Ulcer, necrotic in appearance, on tip of tongue (see Fig. 12); very emaciated. Lungs—"pancreas-like" friable condition in usual situations. Heart—great effusion into pericardium; heart apparently healthy. Lymphatic glands enlarged and reddened in thorax. Abdomen—great effusion into peritoneal sac. No ulcers in intestine; no red enlarged glands about cæcum; no hæmorrhages. Other abdominal organs apparently healthy.

The special points in this case are the necrotic ulcer on the tongue; the presence of a very large amount of fluid in the peritoneal sac without any ulceration or peritonitis; and the history of this case, especially with reference to the origin of the outbreak.

FIG 42.

Same history as in pig 41. First noticed markedly ill on July 13. Since then been going back rapidly. Its lungs appeared to be specially affected, as it had great dyspnœa. Killed July 25.

Post-mortem.—Punctate rash all over the body. Ears are bleeding at tips. Lungs—acute red friable pneumonia, together with acute pleurisy, throughout the lung. Heart—acute pericarditis with adherent pericardium. Lymphatic glands enlarged and reddened throughout the body. Abdomen—very numerous ulcers in colon and cæcum. Kidneys—"turkey egg"; no hæmorrhage in papillæ. Bladder—two large submucous hæmorrhages. Gall-bladder—numerous submucous hæmorrhages.

The special points in this case are its history as in case 41; and the large submucous hæmorrhages in urinary and gall-bladders.

FIG 43.

By this time (Aug. 6) the sows from source D which were in pig, when they arrived at the piggery, had farrowed, and their pigs were about a fortnight old. It was noticed that these young pigs were suffering markedly from diarrhœa. Some of the fæces was removed from the anus of one of the small pigs of sow 2, and plated on M'Conkey lactose plates. The plates after incubation showed a few large lactose fermenters (*B. coli*?), but the great majority of the

organisms present consisted of small pin-point deep acid colonies and a much larger pale colony, which, however, after a few days became red in colour, having meantime formed a little acid: subcultures were made from each of these two latter types of colonies, and their reaction on media is given in Table XI.

Both were gram-negative pleomorphic cocco-bacilli. Both when grown on agar media rapidly died off unless subcultured frequently.

TABLE XI.

	Small red colony.	Large red colony.		Small red colony.	Large red colony.
Litmus milk . . .	O	O	Mannite . . .	O	O
Inuline . . .	O	O	Saccharose . . .	A+g	A+G
Glucose . . .	A+g	A+G	Galactose . . .	A+g	A+G
Dulcite . . .	O	O	Dextrine . . .	A+g	A+G
Lactose, neutral red .	O	O	Sorbite . . .	O	O
Salicine . . .	O	O	Peptone (Indol) .	? +	? +
Maltose . . .	A+g	A+G	Nitrate water (nitrites)	+	+
Adonite . . .	O	O	Gelatine . . .	O	O
Lactose . . .	O	O	Litmus milk (10 days)	A	A
Raffinose . . .	O	O			

Symbols as before.

The special points in this case are the presence of diarrhoea in the small pigs, and the recovery of a special organism practically in pure culture from the fæces.

The points to be noted about the organism are—

(1) The organism occurred on M'Conkey lactose plates in two distinct forms—

(a) Very small deeply acid colony.

(b) Much larger pale colony, which afterwards developed a slight acidity.

(2) (a) and (b) were morphologically and culturally identical.

(3) They further resembled one another in that while both produced acid on lactose M'Conkey plates, neither of them in subculture produced any acid in lactose peptone water.

FIG 44.

Small pig eighteen days old from sow 1. Died on August 11.

Post-mortem.—There were granulating wounds on the side of the face and jaw as if the pig had been tramped on. Lungs—atelectasis of anterior part of right and left lung and of middle lobe of right

lung; no inflammation present. Heart healthy. Abdomen—small intestines slightly congested. Colon and cæcum reddened and injected with hard fæces in them. Umbilicus healthy. Small hæmorrhages into kidney. Other abdominal organs healthy.

Cultures, made from small intestine (none from cæcum) on M'Conkey lactose plates, gave pure culture large acid colonies; no pale colonies present.

FIG 45.

Small pig, same history as pig 44. Died August 11.

Post-mortem.—Lungs quite healthy. Heart—a few hæmorrhages on epicardium. Abdomen—small intestines deeply inflamed. Very numerous petechial hæmorrhages subperitoneally over colon and cæcum; mucosa of cæcum and colon inflamed; hæmorrhages in kidney; enlarged and reddened mesenteric glands.

Cultures.—Small intestine (M'Conkey lactose plates)—pure culture large acid colonies. Colon (M'Conkey lactose plates)—mixture of large red acid colonies and small red acid colonies, as in pig 43. Mesenteric glands—pure extensive culture of small dewdrop colonies. Morphologically the small red colonies from the colon and the organism from the mesenteric glands are identical.

The special point in this case is the presence of this small lactose fermenter in the colon and of an organism, identical with the small dewdrop colony of the large pigs in the lymphatic glands draining the colon area.

FIG 46.

Two days old pig from sow 1. Found dead on August 14.

Post-mortem.—Lungs healthy. Heart ditto. Abdomen—very marked hæmorrhage and inflammation of the mucous coat of colon and cæcum; punctate hæmorrhages in kidneys; mesenteric glands enlarged and reddened.

Cultures from small intestine (on M'Conkey lactose) gave (1) large number of large lactose fermenters; (2) equal number of small lactose fermenters as in colon of pig 45 and fæces of pig 43.

FIG 47.

Pig twenty-two days old from sow 1. Found dead August 15.

Post-mortem.—Covered over, especially on abdomen, with purple hæmorrhagic rash. Lungs healthy. Heart—large quantity of red-stained fluid (containing red blood corpuscles) in pericardial sac. Very numerous subepicardial hæmorrhages all over surface of heart (see Fig. 3). Lymphatic glands enlarged and reddened all through the

body. Abdomen—very numerous subperitoneal hæmorrhages beneath the parietal and visceral peritoneum; large number of submucosal hæmorrhages in stomach, small intestines, and large intestines. Kidney—"turkey egg" appearance.

Cultures.—Heart blood—pure extensive culture of small dewdrops. Spleen, do. do. Lymphatic gland—no growth. Small intestine (on M'Conkey lactose)—mostly large lactose fermenters (*B. coli*?), but considerable number of small pin-point lactose fermenters.

TABLE XII.

	Small acid colony: intestine.	Heart- blood culture.		Small acid colony: intestine.	Heart- blood culture.
Litmus milk . . .	A	A + C	Mannite	O	O
Inuline	O	O	Saccharose . . .	Ag	A
Glucose	A + g	A	Galactose	Ag	A
Dulcite	O	O	Dextrine	Ag	A
Lactose, neutral red.	O	O	Sorbite	O	O
Salicine	O	O	Peptone (Indol) .	+	O
Maltose	O	A	Nitrate water (nitrites)	+	+
Adonite	O	O	Gelatine	O	O
Lactose	O	O	Litmus milk (10 days)	A	A + C
Raffinose	O	O			

Symbols as before. Heart-blood culture here is the culture exactly as it was removed from the heart blood—*i.e.*, reactions are not the reactions of a *single* colony. Spleen culture was dead in twenty-four hours, so that its reactions could not be tested.

FIG 48.

Same history and much the same post-mortem appearances as in pig 47, except that there were not so many hæmorrhages in the abdomen. Died on August 15.

Cultures.—Heart blood—a few colonies of small dewdrop. Pericardial fluid—no growth. Spleen—no growth. Lymphatic gland—thirty colonies small dewdrops. Intestine (M'Conkey lactose)—growth consisted of large lactose fermenters. These cultures (heart blood and lymphatic gland) were made on the 15th August; their appearance was noted after twenty-four hours' incubation on the 16th; tubes were heavily inoculated from them on the 16th; when incubated, however, for twenty-four hours they showed no growth. They thus died out rapidly in artificial culture.

FIG 49 (Sow 2).

Sow from source D and of group δ ; vaccinated twice; was in young when brought to the piggery in April. After it had been vaccinated it was removed from the general piggery, about the beginning of May,

to a new piggery situated at a distance, where no pigs had been before. It was kept there all summer, throve well, and grew very fat. The piggery just mentioned was run as an entirely separate establishment from the other, with a special caretaker to look after it, and there was no communication between the two piggeries. On July 21 this sow gave birth to eight pigs. After this she remained quite well apparently for some time, but began to be dull and off her food about August 8. Her fæces also at this date were noticed to be in the form of hard lumps, tarry in colour, angular in shape, and covered with shreds of mucus. Between August 8 and August 18, when she died, she gradually became worse, developing a bad cough, a hæmorrhagic rash all over the body, and bleeding profusely from the nose at times. Her pigs, meantime, were very badly affected with diarrhoea.

Post-mortem (performed twenty-four hours after death).—Animal very fat; numerous subcutaneous hæmorrhages. Lungs—both sides completely consolidated, deep plum-coloured, and very friable; pleurisy all over; marked hæmorrhages in larynx and trachea. Heart—apparently healthy. Lymphatic glands enlarged and reddened all through body. Abdomen—numerous subperitoneal hæmorrhages all over small intestine. Cæcum and colon—no ulcers; minute petechial hæmorrhages in the submucosa, and one very large one under mucous membrane and bulging into the lumen of the gut.

Cultures.—Although only recently dead, the animal was putrid when it was examined. Consequently no cultures were made.

FIG 50.

Pig about a month old, from sow 3. Killed August 24.

Post-mortem.—Lungs healthy, except for patch of atelectasis. Heart—great excess of pericardial fluid; no hæmorrhages. Lymphatic glands all through body enlarged and reddened. Intestines—congestion.

Cultures.—Heart blood—no growth. No other cultures made.

FIG 51.

History as in pig 50. Killed August 24. Post-mortem as in pig 50.

Cultures.—Heart blood—slight growth of small dewdrop colonies. Lymphatic glands—good growth of same colonies.

FIG 52.

History as in pigs 50 and 51. Killed August 24. Post-mortem as in pigs 50 and 51. Cultures as in pig 51, with, in addition, some staphylococci in gland culture.

FIG 53.

History and post-mortem appearances as in pigs 50, 51, and 52. Cultures: Heart blood—no growth; gland—no growth.

FIG 54.

Same history as in pigs 50, 51, 52, and 53, only this case was much more obviously ill. Killed August 24.

Post-mortem.—Lungs healthy except for patch of atelectasis. Heart—marked hæmorrhages and excess of pericardial fluid. Lymphatic glands enlarged and reddened all over body. Abdomen—cæcum and colon show a diphtheritic exudate along their whole length. When viewed from the outside this exudate is seen as discrete white dots. Beneath the exudate the mucosa is deeply congested and hæmorrhagic (see Figs. 13 and 14). Small intestines deeply congested and inflamed. Stomach—marked hæmorrhages into mucosa. Bladder—hæmorrhages into submucous tissue. Kidney—no hæmorrhages seen.

Cultures.—Heart blood—no growth. Lymphatic glands—pure culture dewdrops. No cultures made from other organs.

FIG 55.

Pig of sow 1, about a month old. Found dead August 24.

Post-mortem.—Lungs—extensive pneumonia of both sides. Heart—extensive acute pericarditis (see Fig. 19). Lymphatic glands all through body enlarged and reddened. Intestines—congestion present.

Cultures.—Heart blood—pure extensive culture of small dewdrop colony. Lungs, do. do. Lymphatic glands—no growth.

Special point in this case is the presence of the pneumonia and the pericarditis.

FIG 56.

Pig from source D and of group γ ; not vaccinated. This pig appeared fairly well till July 10, when it was noticed to be dull. On one occasion while it was eating it suddenly tumbled down on the floor on its right side and lay kicking. After a few minutes it was helped to its feet, but again tumbled down on its right side. After some time it got up by itself. On its attempting to walk, it was noticed that it leaned over to the right side and its right limbs appeared to be somewhat stiff. Next day it appeared to be quite well again, and remained comparatively well till July 25, when it was noticed that it was beginning to desquamate in large scales all over the back of the body. Behind both ears there was also present large patches covered with moist scales, and resembling a weeping intertriginous eczema. On July 29 it was noticed that when the animal raised its head in the air the skin of the back was thrown into heavy transverse folds, giving it a pachydermatous appearance. From August 3 till August 24 (when it was killed) it was out of sorts, being dull, not eating its food, and having a discharge from the eyes and nose, its eyelids being gummed together in the mornings. Meantime the skin condition had improved greatly.

Post-mortem (August 24).—Lungs—"pancreas-like condition" in usual places. Enlarged and reddened lymphatic glands at root of lungs. Heart apparently healthy. Intestines—no signs of disease.

The special points in this case are the presence of the peculiar nervous attack, and the scaly skin condition.

FIG 57.

Pig from source A and of group γ ; had been vaccinated. It had never been noticed ill till about August 8, when it was noticed to be lame in hind quarters. It continued, however, to eat its food. The lameness, however, continued, and became worse, till on August 12 it was noted that it was very unsteady on its hind legs. A slight rain had made the concrete floor of its court slightly damp, and it was noticed that it could hardly walk on it. While it was standing eating at its trough it was pushed gently on the shoulder with the point of an umbrella, when it immediately sat down on its hind quarters like a dog and remained sitting for some time. From August 12 till August 24, when it was killed, the lameness improved, but the animal still remained dull and out of sorts.

Post-mortem.—Nothing abnormal found in any part of the body, nor was any disease in the joints found which could account for the lameness of the hind quarters.

The special point to be noted in this case is the transitory weakness in the hind quarters.

FIG 58.

Pig from source A and of group γ ; had been vaccinated. It had been a pen-mate of pig 57. It had appeared quite well up to August 4, on which date it was noticed to be dull and not taking its food. On August 7 it was noticed to be lame in the left hind leg. The leg was not painful to the touch, as it could be handled quite freely. There was a small pinkish rash all over the body. The animal was taking its food. On August 8 the animal was noticed to be paralysed in both hind legs. The paralysis was so marked that, when the animal wanted to come from its pen to the trough to feed, it progressed entirely on its fore legs and trailed its hind quarters behind it on the ground. From August 8 till August 24, when it was killed, the paralysis continued, though it improved considerably towards the latter part of this time. All the time it continued to eat its food quite well, trailing itself to the trough to do so.

Post-mortem.—Nothing abnormal found in any part of the body.

The special point in this case is the presence of the very marked though transitory paralysis.

FIG 59.

This pig and the following four pigs, though not *in extremis*, were killed because they had diarrhoea and were not thriving. They were about five weeks old, and came from sow 1. They were killed on August 28.

Post-mortem of pig 59.—Lungs—healthy; no atelectasis. Heart—no disease or hæmorrhages. Lymphatic glands—no enlargement or reddening. Abdomen—small intestines very inflamed. Cæcum and colon very inflamed, with ulcers and diffuse fine diphtheritic exudate on surface.

Cultures.—Heart blood—pure extensive culture small dewdrops. M'Conkey lactose plate from cæcum gave 50 large acid colonies and 50 small pin-head colonies, deeply acid in tint.

FIG 60.

History and post-mortem as in pig 59.

Cultures.—Heart blood—pure extensive culture of small dewdrops. M'Conkey lactose plate from cæcum gave 50 large acid colonies, 45 small pin-head deeply acid colonies, and 5 medium-sized pale colonies, which after two days at room temperature became faintly but distinctly acid.

FIG 61.

History and post-mortem as in pigs 59 and 60.

Cultures.—Heart blood—no growth. M'Conkey lactose plate from cæcum gave 40 large acid colonies, 50 small pin-head deeply acid colonies, and 30 medium-sized pale colonies, as in pig 60.

FIG 62.

History and post-mortem as in pigs 59, 60, and 61.

Cultures.—Heart blood—a single colony of a large coliform organism. M'Conkey lactose plate from cæcum gave 50 large acid colonies, 30 small pin-head deeply acid colonies, and 2 medium-sized pale colonies, as in pigs 60 and 61.

FIG 63.

History and post-mortem as in pigs 59, 60, 61, and 62.

Cultures.—Heart blood—no growth. M'Conkey lactose plate from cæcum gave 50 large acid colonies, 20 small pin-head deeply acid colonies, 1 pale colony, medium-sized, as in the others.

FIG 64.

History much the same as in pigs 59, 60, 61, 62, and 63, except that this pig came from sow 6.

Post-mortem.—Same as in these pigs, except that in this case there was a patch of atelectasis in the lung.

Cultures.—Heart blood gave pure extensive culture small dewdrop. M'Conkey lactose plate from cæcum gave 50 large acid colonies, 40 small pin-head deeply acid colonies, 10 medium-sized pale colonies as above.

The following are the reactions and characters, &c., of some of these organisms obtained from pigs 59-64:—

The large acid colonies were not tested or examined further.

The cultures from the heart blood of the cases where such were obtained, as also the small pin-head acid colonies and the medium-sized pale colonies from the M'Conkey lactose agar, were examined with the following results:—

Heart cultures—

Pure extensive cultures of a small dewdrop colony were obtained in cases 59, 60, and 64.

After twenty-four hours these cultures were examined microscopically, and showed the presence of numerous gram-negative, pleomorphic, polar-staining cocco-bacilli.

Subcultures were at once made by heavy inoculation on ordinary agar from all three. After incubation for twenty-four hours at 37° C., a growth was obtained only in the case of pig 64, the others having meantime died. The reactions of these organisms on the sugars, &c., will be given later.

M'Conkey lactose plates from cæcum—

Numerous subcultures were made on agar from the "small acid" and "pale colonies" derived from all the six cases.

The agar subcultures from the small acid colonies after twenty-four hours' incubation showed a discrete growth of a fairly large-sized colony—about three times the size of the dewdrop. The agar subcultures from the pale colonies, however, gave a discrete growth of a mixture of dewdrop colonies and of larger colonies, such as has just been described in the case of the small acid colonies.

Subcultures on agar were now made from these last described dewdrop colonies (pale colony source) in the cases of pigs 62 and 64. After twenty-four hours at 37° C., there was a discrete growth of colonies of the larger variety only, no dewdrops being present. At the same time a "large" colony from the same "pale colony" source was subcultured on agar in the case of pig 62.

All these organisms were gram-negative, pleomorphic, bipolar cocco-bacilli.

Further, all these organisms required repeated subculture, otherwise they died out. If they were left for a few days, and a subculture was then made with heavy inoculation, either a few isolated colonies grew or nothing at all.

TABLE XIII.—REACTIONS OF HEART AND CÆCUM ORGANISMS FROM FIGS 59-64.

	Small red colony, Fig. 60.	Small red colony, Fig. 61.	Small red colony, Fig. 62.	Small red colony, Fig. 64.	Pale colony, Fig 61.	Large colony from agar subculture of pale colony, Fig 62.	Dewdrop colony from agar subculture of pale colony, Fig 62.	Dewdrop colony from agar subculture of pale colony of Fig 64.	Heart-blood culture of Fig 64.
Litmus milk . . .	A ?	A ?	A ?	A ?	A ?	A ?	A ?	A ?	A + C
Inuline	O	O	O	O	O	O	O	O	O
Glucose	A + G	A	A	A	A	A	A	A	A
Dulcitate	O	O	O	O	O	O	O	O	O
Lactose, neutral red	O	O	O	O	O	O	O	O	O
Salicine	O	O	O	O	O	O	O	O	O
Maltose	A + g	A ?	A ?	O	O	A	O	O	O
Adonite	O	O	O	O	O	O	O	O	O
Lactose	O	O	O	O	O	O	O	O	O
Raffinose	A ?	A ?	A	O	A ?	A ?	O	O	O
Mannite	O	A	A	A	A	A	A	A	O
Saccharose	A + G	A	A	A	A	A	A	A	O
Galactose	A + G	A	A	A	A	A	A	A	O
Dextrine	A + G	A	A	A	A	A	A	A	O
Sorbite	O	A	O	O	O	O	O	O	O
Peptone (Indol) . .	?	?	?	?	?	?	?	?	?
Nitrate water (nitrites)	+	+	+	+	+	+	+	+	+
Gelatine	O	O	O	O	O	O	O	O	O
Litmus milk (10 days)	Acid	Acid	Acid	Acid	Acid	Acid	Acid	Acid	A + C
	1	2	3	4	5	6	7	8	9

Symbols as before.

The special points in connection with these organisms are—

- (1) Small red colony and pale colony from cæcum appear from their reactions on the various media to be the same organism.
- (2) Small red colony and pale colony, although they produce acid on M'Conkey lactose media, do not produce acid in lactose peptone water.

- (3) The variation in size of the colonies from one agar culture to another, as exemplified in the case of pigs 62 and 64.
- (4) The variations in the fermentative activities qualitatively, as seen in Table XIII., and quantitatively, as especially seen in organism No. 1.
- (5) The reactions obtained in the organisms in Table XIII. should be compared with those obtained in Tables XI. and XII. from similar cases.

PIGS 65, 66, 67, 68, 69, 70, 71, 72.

These were small pigs about five weeks old, obtained from sows 6 and 3. They were not thriving, and diarrhoea was present in most of them. They were killed on August 29.

Post-mortems.—There was no discharge from the eyes or nose of any of them. Lungs healthy, not even atelectasis, except in one case where there was consolidation of right middle lobe and anterior part of right posterior lobe. Heart—three cases showed great excess of amber-coloured fluid; hæmorrhages were present in the heart of one case. Lymphatic glands—generally no enlargement or reddening; in the case, however, where the lungs were pneumonic, there were enlarged and reddened glands all through the body, especially in the mesentery and thorax. Abdomen—the cæca and colons of all these pigs were markedly inflamed and congested, without, however, ulcers or diphtheritic exudate. *Cultures.*—No cultures were made from these cases.

PIGS 73, 74, 75.

These were the remaining pigs from sow 3. They had been the best thriving of all the pigs, but latterly they had begun to go back, with diarrhoea, dulness, lack of appetite, discharge from eyes and nose, coldness of skin, and very deep blue colour of the ears. These pigs were killed on August 30, but it was not possible to do a post-mortem on them.

Special points to be noted in connection with these small pigs—viz., 43-48, 50-55, 59-75:—

- (1) They were obviously affected with a contagious disease.
- (2) As they had not been in contact with any other pigs, the presumption is that they got this disease from their mothers.
- (3) The chief symptom of this disease was diarrhoea; and the chief post-mortem appearance found was hæmor-

rhagic and diphtheritic inflammation of the intestines, especially of the large intestine.

- (4) Although the symptoms and post-mortem appearances just described were those manifested in the most of the cases, several of the older-standing cases showed the transition stages to the condition found in the pigs of group β . Thus many of them showed enlargement and reddening of the lymphatic glands throughout the body, while hæmorrhages throughout the body were observed in several cases. Effusion, blood-stained and otherwise, into the pericardium was also noted in quite a few cases; extensive pneumonia was found in case 55 and one other case; while acute pericarditis was found in case 55; definite discrete ulcer formation in the colon was found in several cases.

There next fall to be described two groups of pigs whose history has a special bearing on the epidemiology of the disease—

- (1) A group of ten pigs of group β , *which were not vaccinated*. These are numbered pigs 76-85. Pig 17, already described, is also one of this group.
- (2) The six sows from source D and of group δ , which were in young when they arrived at the piggery.

PIGS 76-85 (with Pig 17).

These pigs came in a batch of thirty-seven from source C about the beginning of May. When they arrived they were about two months old. They had not been vaccinated. They remained quite well till about May 19, when they were noticed to be discharging from the eyes and nose. At this time the same symptoms were being exhibited by the other members of group β , which had been vaccinated but in a very much more severe degree. Their temperatures ranged from 103.6° F. to 105.6° F. No diarrhoea was present. Several of them showed the large dark spots on the skin already described for the vaccinated members of the group. The pigs were never severely incapacitated, and ate their food practically the whole time. They were also quite alert and showed no evidence of dulness. The majority of them were practically better within a week. (One of them, however—pig 17—was more seriously affected and had to be killed on June 19.) From about May 26 till July 31 the ten pigs thrived apace, and at the end of this period were about

twice the size of their vaccinated fellows, among which the disease continued unabated. From July 31 to August 4, a sharp illness, characterised by formation of wheals and disinclination to eat, attacked them all. This condition rapidly passed off and the animals began again to thrive markedly.

The special points to be noted in connection with this group are that these pigs were unvaccinated; the mildness of the disease in their case; the thriving condition of the pigs prior to and subsequent to the disease, as evidenced by the fact that at the end of July they were about twice the size of their fellows which had been vaccinated.

Sows 1, 2, 3, 4, 5, 6.

These sows came from source D about the end of April. They were in young when they arrived. They were vaccinated twice in the beginning of May, and were then removed to a newly constructed wooden piggery on ground where no pigs had been previously kept, and at a considerable distance from the other piggery. This new piggery was run as an entirely separate establishment, there being no communication between this place and the old one. It should be noted, however, that previous to their removal they had been in various parts of the old piggery; and further, that the disease had broken out there before their removal.

Sow 5. On the 16th June sow 5 was dull and refused to take her food. She remained in much the same condition till the 19th June, when she began to recover, and was all right again by the 22nd of June. This sow farrowed on July 23. It was very ill on the morning of July 25, and all its pigs were dead except one, which was transferred to sow 2, which had farrowed that day. Sow 5 was all right again on July 27.

Sow 6. On 27th June sow 6 became out of sorts, dull, and refused food. On the 5th of July it became very definitely ill, and remained in this condition till the 16th July, during which time it was never seen to take any food. After this it gradually got better. This sow farrowed on August 14, giving birth to numerous macerated foetuses, and also to one live pig, which, however, was puny and ill developed.

Sow 3 was slightly but noticeably ill from about June 27 to June 30, but soon recovered. It gave birth to eight pigs on July 25.

Sow 4 was also slightly but noticeably ill from June 27 to June 30, but soon recovered. It gave birth to a large number of pigs on July 29, but killed them all, except three, by overlaying.

Sows 1 and 2 showed no signs of illness previous to pigging.

On July 31 sow 1 gave birth to ten pigs. On August 3 she was noticeably ill, being dull and not feeding. On August 5 she was very much worse, lying down listless and dull and paying no heed

to her young. On the 7th it was noticed that her fæces were in the form of large, tarry, hard, angular lumps covered with threads of mucus. After this she rapidly improved, and appeared all right again about the 9th of August.

The history of sow 2 has already been described under pig 49.

Special points in these cases are—

- (1) The acute though ephemeral nature of the disease in some of these cases.
- (2) The result of the disease on pregnancy as seen in the case of sow 6.
- (3) The intestinal nature of the disease in some of these cases, as evidenced by the nature of the fæces in sows 1 and 2.
- (4) The occurrence of a disease among their pigs characterised by diarrhoea and marked intestinal lesions.

SYMPTOMATOLOGY EXHIBITED IN THESE EPIDEMICS.

With the exception of pig 6 and the various sows 1-6, with their suckling pigs, all the animals exhibited more or less indefinite symptoms, characteristic rather of a subacute or chronic indefinite affection than of an acute disease of one single type. Moreover, in a single case, the disease symptoms varied from time to time. Thus a pig would be apparently well at one time, would then be ill for a short time, and would then appear to be more or less well for some time, only to fall ill again.

General symptoms.—When a pig was ill it generally showed it by a dulness, listlessness, and lack of appetite, and a desire to stay inside and not come out. In many cases these were all the symptoms observed.

Abdominal symptoms.—Except in the case of the epidemic among the small pigs, about one month old, which all showed this symptom, diarrhoea was the exception. It was observed in one case only—pig 15—and that for a very short time. Instead of diarrhoea there was rather constipation, and in certain cases, such as pig 39 and sows 1 and 2, there was the passage of hard, angular, sticky, tarry masses covered with threads of mucus.

Lung symptoms. — Coughing was a very prominent symptom. This symptom occurred among all the ages of pigs from two months upwards, and whether the animal appeared ill or not. Especially noteworthy was its occurrence among the older pigs which were eating their food well, and which were apparently quite healthy, as it was indicative, as post-mortems on some of them showed, of a persistence of a disease condition in the lungs. In such cases this symptom was especially noticeable after the pigs had been made to exert themselves, as in running. The cough was a soft open-mouthed one. Laboured breathing, with heaving sides, occurred chiefly in the more serious cases, and was indicative of gross lung changes, which condition was sometimes associated with pericarditis or great effusion into the pericardial sac.

Circulatory symptoms exhibited were breathlessness, coldness of the body, and blueness of the tips of the ears in the gravely ill cases.

Thirst was very marked, especially in the cases where the lungs were acutely affected, and where there was high fever.

Temperature.—In the more acute cases the temperature was usually raised. The following are the temperatures of twenty-seven pigs when they were affected with the syndrome of symptoms to be described immediately (rash, discharge from eyes and nose, and cough): 103·8; 104·1; 104·2; 103·7; 104·1; 103·8; 103·7; 104·2; 104·0; 104·6; 105·0; 104·0; 103·6; 105·6; 105·2; 104·6; 104·8; 106·0; 104·8; 104·8; 105·6; 104·3; 104·6; 103·6; 104·9; 103·3; 103·0.

Nervous symptoms.—The nervous seizure in pig 56 should be noted. It is an open question whether this may not have been of the nature of a heart seizure. A much more common nervous symptom exhibited, however, was definite paralysis of one or more limbs or of the hind quarters. This symptom was observed in pigs 4, 15, 57, and 58. In pig 4, the paralysis was definitely associated with the presence of changes in the grey matter of the spinal cord (see Fig. 20). In pigs 57, 58, and 15, the symptom, after having been present to a marked degree, passed off and the animal recovered. Pig 58 is especially noteworthy in this connection, as there could be no doubt of the presence here of a definite paralysis. The

paralysed limbs in all these cases could be handled quite freely, and there was no evidence in any of them of a local condition to account for the symptom. It is noteworthy, too, that this symptom, except in the case of pig 4, occurred among the older pigs; and that the pigs affected appeared to be otherwise in fairly good health, and continued to take their food. One of them habitually trailed its hind quarters when trying to reach the food trough. One pig showed lameness due to an abscess in the foot.

Special syndrome.—A special syndrome of symptoms was observed in the more acute cases among the pigs aged from 2-4 months. This consisted of discharge from the eyes and nose (see Fig. 1), sneezing, coughing, fever, thirst, difficulty in breathing, dulness, inappetence, retardation of growth, and the presence all over the body of a raised macular rash, deep red in colour, and of the size of a shilling piece (see Fig. 2). Over each spot at times there was a slight exudation together with the presence of scales. The spot did not disappear on pressure. Discharge from the eye and nose was observed in a few of the older pigs, from 4-6 months; and again, in the group of suckling pigs reared in August, some of the older ones were beginning to manifest this symptom when they were about one and a half months old.

Skin eruptions.—The eruption met with in the special syndrome has already been mentioned. Other skin eruptions were, however, frequently observed. Thus pig 6 showed an extensive reddish eruption all over the under surface of the body, which at first disappeared on pressure. This led one to think (as this was the first case to occur) of swine erysipelas. As the disease progressed, the eruption became definitely hæmorrhagic in this case.

A very large number of the more acute cases exhibited petechial or macular hæmorrhagic rashes on various parts of the skin.

The blueness of the tips of the ears and the coldness of the body surface, due in all probability to weakness of the circulation, has already been alluded to.

Two pigs exhibited a generalised desquamative hypertrophic condition of the skin. The skin had a pachydermatous appearance, and there was a moist weeping intertriginous

eczematous condition between the backs of the ears and the adjacent body surface. In some of the pigs over six months old a curious, circular, black-coloured and flat eruption appeared in the middle line of the body beneath the chin and in the axillary and inguinal regions. It consisted of isolated spots about the size of a penny, deep black in colour, and flush with the surface. This eruption lasted for a very short time only, and there appeared to be no constitutional symptoms.

All the unvaccinated pigs of age from 2-4 months, some time after they had thrown off the syndrome above mentioned, came out in large wheals all over the body. The general health of the animals was not much affected by this eruption, which rapidly disappeared. It was possibly due to some error in diet.

Lack of growth.—This was very noticeable. With the exception of the unvaccinated pigs of group β (age 2-4 months) it may be said that the pigs of group β and γ were only half the size they should have been for their age.

The symptoms exhibited may be roughly arranged according to their occurrence in the age groups as follows:—

Group α ($\frac{1}{2}$ -1 $\frac{1}{2}$ months). Diarrhœa, and towards the end of this time discharge from the eyes and nose.

Group β (2-4 months). Lung symptoms and discharge from eyes and nose.

Group γ (4-6 months). Modified lung symptoms without discharge from eyes and nose; presence of paralysis.

Group δ (6 months and over). Loose infrequent cough and slight want of thriving.

The lingering, chronic, in some cases almost unobservable character of the symptoms exhibited in the second outbreak in Piggery II. should be specially noted in reference to—

(1) The liability of the disease to escape recognition as swine fever, the animals being considered to be wasters or piners, or the disease being considered to be the so-called swine plague, so-called enzootic pneumonia of pigs, &c.

(2) The liability of these animals, especially those less observably ill, continuing the disease in a piggery or

carrying it to another piggery, after having been transferred there under the assumption that they were healthy.

PATHOLOGICAL FINDINGS IN THESE CASES.

Intestines.—The *small intestine*, especially in the pigs under 4 months, was often acutely and hæmorrhagically inflamed. Much more frequently, however, the *large intestine* was involved. Here, especially in the young pigs under 1½ months, but also in some of the older pigs, such as pig 39, there was acute hæmorrhagic congestion, accompanied sometimes by a more or less uniform whitish deposit on the surface. Ulcer formation, especially round about the ileocæcal valve, occurred in a very large number of cases. In some cases the ulcers were acute, in others chronic. They were chiefly found in pigs over 2 months old, the acute ulcers being chiefly found in pigs under 4 months and the chronic ones in pigs over this age. In the acute ulcers, the subjacent peritoneum was often affected, giving rise to a localised peritonitis.

Lungs.—In a very large number of cases there was present a grey pancreas-like consolidation of the right middle lobe, the postcardial lobe, and the lower and anterior borders of the right and left lung. Histologically the consolidation was of the nature of a catarrhal broncho-pneumonia. There was no evidence of helminthiasis or tuberculosis. This condition was rare in pigs under 1½ months, occurring as it did in only two cases. Its occurrence was at its maximum, in numbers affected, extent, acuteness and gravity of the lesions, in pigs from 2-4 months, where in addition there was often an associated pleurisy. Above the age of 4 months this lesion began to diminish in frequency and severity, until in pigs of 6 months and over it was absent, or consisted of a very small patch of almost entirely resolved pneumonia.

Heart.—In many cases, especially of the more acute types, there was a great effusion of clear straw-coloured fluid into the pericardial sac. In some cases this fluid was deeply red in colour, the colour being due to red blood corpuscles

derived from subepicardial hæmorrhages—a pathological change found in a great number of cases. These subepicardial hæmorrhages, as also the effusion, occurred chiefly among the pigs from 1-4 months old.

In some cases there was acute pericarditis. This usually occurred in association with a pneumonia, and was present chiefly in the pigs from 1-4 months old. Chronic pericarditis, with adherent pericardium, no longer in an active condition, was found in two cases—one a pig between 2 and 4 months old, and one a pig over 6 months old.

Spleen was found enlarged in only one or at most two cases.

Effusion into the peritoneum.—In a considerable proportion of cases, between the ages of 2 and 4 months, a very large accumulation of clear fluid with coagula was found in the peritoneal sac. This was sometimes, but not always, associated with a local peritonitis over acute ulcers in the large intestine. In most cases its origin may be assumed to be dropsical and of the same nature as the clear fluid already mentioned as occurring in the pericardial sac.

Hæmorrhages.—It cannot be too strongly emphasised, however, that hæmorrhage was the all-important pathological change found in the above cases. Thus hæmorrhages occurred in the lymphatic glands (see Fig. 9), giving rise to the reddening and enlargement; in the heart in the myocardial and subepicardial tissues, and giving rise to the blood-stained appearance of the effusion into the sac (see Fig. 3); into the lungs (see Fig. 7); into the pleura; into the subperitoneal tissue; into the skin (giving rise to some of the rashes); into the subepithelial tissues of the tongue; into the subepithelial tissues of the stomach, small intestine, and large intestine (see Fig. 8); into the spleen; into the substance of the liver; into the kidney substance (giving rise to the “turkey egg” appearance); into the papillæ of the kidney (Fig. 6); into the submucous tissue of the bladder (Figs. 4 and 5); into the submucous tissue of the gall-bladder; into the muscles, &c., &c.

The view, further, is expressed here that all the gross pathological lesions (except possibly the pneumonia) found in the above cases are referable to hæmorrhages having

taken place in various situations during the more acute stages; and according to the size of these and the situation where they have occurred, would arise the various pathological lesions found in the disease.

If the hæmorrhages are small and punctate, or of a much larger size, and occur in the body of an organ such as the kidney, spleen, liver, lymphatic glands, heart, or on a moist surface not liable to bacterial contamination, or on a surface which, though liable to bacterial contamination, is habitually dry, then the hæmorrhage is apparently resolved in the ordinary way. If, however, these hæmorrhages are small and occur on a moist surface liable to bacterial contamination, and more especially if they can cut off by mechanical pressure the blood supply to the more distal parts, then it would appear that superficial necrosis and surface hæmorrhage takes place. This is what would appear to occur in the small intestine, and especially in the large intestine where hæmorrhages take place between the glands of the mucosa towards their bases, causing necrosis of the superficial layers of the mucosa (see Figs. 13 and 14).

On the other hand, when the hæmorrhages are large, and occur on surfaces such as have been mentioned, the changes produced may be observed by studying them as they occur—

1st, in the bladder.

2nd, in the skin generally.

3rd, on the moist skin of the nose.

4th, on the skin of the tongue and mouth.

5th, on the mucous membrane of the large intestine.

1. In the bladder. Fig. 4 is a photograph of the bladder of a pig, showing there numerous and large hæmorrhages. Fig. 5 is a section through the bladder wall. The hæmorrhage comes right up to the mucous membrane, and the red blood cells may be seen penetrating in between the epithelial cells themselves. There is no sign of ulceration or necrosis here. The reason may be that there are no organisms or secretion products present to turn the balance in favour of necrosis of the epithelium, or that the collateral circulation may be sufficient to keep the mucosa intact. It is, however, difficult to see how this can be (as the hæmorrhage is infiltrating between the epithelial cells of the mucosa, and is

actually lifting the mucous membrane up from the sub-mucosa), unless the large hæmorrhages may be looked on as occurring in sinuses, in which case a pseudo-circulation may be kept up owing to the filling and emptying of the bladder.

2. On the skin generally, as in the syndrome described under the symptomatology, where large hæmorrhages appeared beneath the skin and disappeared, leaving no ulceration.

3. On the moist skin of the nose. Figs 10 and 11 are from sections from such a hæmorrhage occurring in a case where there were the large hæmorrhages just described on the skin generally. Here there is an absence of inflammatory reaction, such as leucocyte invasion; the changes are those of necrosis; there is a line separating the more or less healthy tissue from the necrosed tissue, and the difference between the healthy and the necrosed epithelium is very obvious. Immediately below the necrosed epithelium is an area of extravasated blood which is in the process of alteration (necrobiosis).

4. On the skin of the tongue and mouth. Fig. 12 is from a case where ulcers of the typical swine fever variety, and hæmorrhages, large enough to bulge the epithelium, were present in equal numbers on the tongue and on the fauces. In the ulcer the following points will be noted—namely, that there is an absence of leucocytic emigration; the condition is a necrotic one; there is a sharp line of demarcation between healthy and necrosed tissue, and an absence of granulation tissue; there is the presence of organising clots in the vessels immediately subjacent to the line of demarcation.

5. Mucous membrane of the large intestine. Photographs are given of sections of a hæmorrhage (see Fig. 8), and ulcer (see Figs. 15 and 17), from a case where these were found together abundantly in one case. As regards the ulcer, the following points will be seen: that there is an absence of leucocytic emigration; the condition is not an inflammatory one; there is the presence of a large necrotic area containing masses of red blood cells undergoing necrobiosis separated from the healthy area by a line of demarcation; there is an absence of granulation tissue and the presence of organising

thrombi in the blood-vessels on the proximal side of the line of demarcation.

The section of the hæmorrhage shows a large effusion of blood into the submucosa. The hæmorrhage was a fairly large one, and the bulge on the mucous membrane at this point as seen in the photograph is due to its presence to a large extent.

With these facts before us, and in the absence of any other feasible explanation, it does not seem too much to assume—

- (1) That hæmorrhage is at the basis of formation of the ulcers regarded as characteristic of swine fever.
- (2) That some factor or factors absent in the case of hæmorrhages into the bladder and skin generally, and present in hæmorrhages into the skin of the nose, of the tongue and fauces, and of the intestine, is the determinant factor in the formation of the ulcer.

Whether this factor is (in the case of the intestine, &c.) the presence of organisms, intestinal secretions, &c., and the absence of free blood anastomosis, it is impossible to say definitely. Certainly the peculiar shape of the intestinal ulcers would be best explained by a hæmorrhage in the terminal arterial supplying a definite almost geometric portion of the mucosa.

In this connection, the geometrical appearance of the large infarcts so common in the intestine when branches of the mesenteric arteries are blocked should be borne in mind. Further, a study of Fig. 18, which represents diagrammatically the course of the arteries from the submucosa to the mucosa in the large intestine, would appear to show that if a hæmorrhage occurred which blocked one or more of these vessels at or about the point *a*, there would be interference with the blood supply to a certain definite area of the mucosa, as the branches from *a* are more or less terminal in nature. With regard to the hæmorrhages generally, they would appear to be toxic in character, caused possibly by an action on the endothelium of the blood-vessels. In this connection the fewness of the organisms, or their entire absence at times in the enlarged and reddened lymphatic glands, is worthy of note.

It would appear that the organising clots found at the base of the ulcers (see Figs. 16 and 17) are the result of the

hæmorrhage rather than the cause of it. They serve to close up the surface after the slough has been separated.

The blueness of the ears and coldness of the body, the hydropericardium, and the peritoneal effusion would appear to indicate a great weakening of the circulation, probably due in most cases to the action of a toxin, but in some cases, no doubt, aggravated by the increased work in the pulmonary circulation due to the consolidation.

In the case of pig 4, which exhibited paralysis, sections of the cord showed the presence of chromatolysis in the cells of the anterior horn of the lumbar region, as also infiltration of the vessel walls with small round cells (see Fig. 20). As the distemper organism was present in the consolidated lung of this case, it is possible that the condition here shown was attributable to the action of this organism. Similar changes have been shown by Dawson and myself¹ to occur in the cords of dogs affected with distemper paralysis.

DISTRIBUTION AND CHARACTER OF THE LESIONS ACCORDING TO THE AGES OF THE PIGS.

Group *a* (pigs under 1½ months). Acute intestinal changes, especially in the large intestine, of the nature of an acute hæmorrhagic, often diphtheritic, inflammation affecting the whole mucosa. Transition to the next group is made easy by the occurrence here of isolated cases of—

- (1) solitary ulcer formation;
- (2) hæmorrhages into the heart, with effusion into the pericardium;
- (3) acute extensive pneumonia with pericarditis.

Group *β* (2-4 months). Here the characteristic changes were acute ulceration of the large intestine, localised peritonitis over these ulcers, and great effusion into the peritoneal cavity; and also extensive acute pneumonia, associated in many cases with hæmorrhages in the heart, effusion into the pericardium, or acute pericarditis.

Group *γ* (4-6 months). The characteristics of group *β* were found here also, but in a much less acute form. The

¹ M'Gowan and Dawson. Journ. Path. and Bact., vol. xix. p. 182, Oct. 1914.

ulcerations were old standing, in some cases healed or healing. There was absence of peritonitis, and the pneumonic condition in the lungs was much less acute, much less extensive, and in many cases in a resolving condition. Pericardial effusion or pericarditis was absent.

Group δ (above 6 months). Here the ulcers were few in number and practically healed. In the lungs the pneumonic patches were evanescent. In some cases no evidence of any disease could be found.

BACTERIOLOGY.

Dealing first with the cases from the second epidemic in Piggery II., it may be seen that, with the exception of pigs 73, 74, and 75, post-mortems were done in all the cases that either died or were killed. At the same time a bacteriological examination was also made in as many cases as possible. It was impracticable to do this latter operation in many cases, while in other cases there were no active lesions to make it worth while doing. In connection with the statement just made relative to the activity of the lesions found, it may be mentioned that the cultures which were taken were as a rule made from active lesions only.

In making a general survey of the results obtained from these cultures, it emerged that there was present in a very large number of cases an organism having the following characteristics: (1) gram-negative; (2) cocco-bacillus, pleomorphic and polar-staining; (3) forming small dewdrop colonies; (4) rapidly dying out even if subcultured frequently at short intervals; (5) giving a different type of growth at different times on different samples of agar, possibly in response to slight variation in reaction, to which this organism was very susceptible.

An organism having these characteristics was found in a very large number of cases in practically pure uncontaminated massive culture. For the purposes of the table to be given immediately, when this occurred the cases in which it did will be recorded as positive, as also will be the cases in group α , where the organism was found in the intestine to an equal or greater extent than the *Bacillus coli*.

TABLES SHOWING POSITIVE RESULTS, AS JUST DEFINED, IN CULTURES MADE FROM THE VARIOUS ORGANS FROM ANIMALS OF THE VARIOUS AGE GROUPS.

AGE GROUP α (up to $1\frac{1}{2}$ months).

Number of cases = 29 ; cultures made in 17.

	Cultures made in	Positive in	No growth in	Not done in
Lung	1 case	1 case	0 case	28 cases
Heart blood	13 cases	7 cases	5 cases	16 „
Lymphatic glands	7 „	4 „	2 „	22 „
Intestine	12 „	11 „	0 case	17 „

AGE GROUP β (2-4 months).

Number of cases = 21 ; cultures made in 12.

	Cultures made in	Positive in	No growth in	Not done in
Lung	12 cases	10 cases	2 cases	9 cases
Heart blood	0 case	—	—	21 „
Lymphatic glands	1 „	1 case	—	20 „
Intestines	0 „	—	—	21 „

AGE GROUP γ (4-6 months).

Number of cases = 14 ; cultures made in 3.

	Cultures made in	Positive in	No growth in	Not done in
Lung	3 cases	2 cases	1 case	11 cases
Lymphatic gland	1 case	0 case	0 „	13 „

AGE GROUP δ (above 6 months).

Number of cases = 7 ; no cultures made.

This Table shows—

(1) The great prevalence of this organism, especially in the acute stages of the disease. The frequency may be even higher than is shown in the positive column, for, owing to the

delicacy of the organism, some of the organs from which cultures were made, and which were noted as showing no growth, may quite well have contained the organism.

(2) This table is derived from a consideration of cultures made from acute lesions, and it will be seen that the presence of this organism in various sites allows of an age table being constructed, comparable with the age table for the pathological findings and symptomatology as follows:—

Age group α (up to $1\frac{1}{2}$ months). This organism is very abundantly present in the intestine, especially in the large intestine, corresponding with the acute inflammation of this part in these cases and with the diarrrhœa. In these cases, however, it is also found in a considerable number of cases in the heart blood and lymphatic glands, and it was also found in one of the cases of pneumonia. These latter cases bridge over the gap to age group β .

Age group β (2-4 months). Corresponding with the predominant lung changes and symptoms, the organism is found in large numbers in the lung cultures. If the blood had been examined more frequently the organism would have been found there in all probability in a large percentage of cases. It was found in the one lymphatic gland examined. No intestinal contents were examined.

In age groups γ and δ the pathological changes are becoming more chronic, as are also the symptoms. Here, however, the organism is found in the tissue which is mainly affected, the lung, and which is giving rise to the chief symptom—namely, coughing.

The next point to be looked at is the further reactions of the organism just mentioned. In this connection there may be considered the organisms of the same kind obtained from pigs 1-5. Before, however, going on to this, it will be necessary to make some remarks with reference to other organisms found.

The bacillus of distemper (*vide supra*) was found in two cases—viz., pig 4, in pure uncontaminated extensive culture from the consolidated lung; and in pig 5, in conjunction with the organism just mentioned. The reactions of these two

strains of the distemper bacillus are given in Table II. (No. 1) and Table III. (No. 2).

This distemper organism was looked for in all the cases that occurred subsequently, and was never found. It should especially be mentioned that cultures were made from the noses of the thirty-seven small pigs of group β , when they were suffering from discharges from the eyes and nose, and in no case was this organism found.

The *Bacillus suipestifer* (classical type) was examined for in every culture made from pigs in this investigation. Organisms resembling it were found in two cases only—namely, pig 1, duodenum; and pig 4 in heart blood, duodenum, and intestine. The reactions of these organisms are given in Table I. (Nos. 10-13) and Table II. (Nos. 2-7). Particular attention, however, is directed to the varying reactions given by these organisms, especially noticeable in Table I., and more especially with reference to organisms Nos. 12 and 13.

Having disposed of these two groups, there only remain to be considered organisms belonging to the group whose characters were defined above. It may be stated again, that all the organisms left in the tables after consideration of the distemper and *suipestifer* group belong to this group.

The question now arises whether these organisms have more common characters, elicited by further tests, than are embodied in the above definition.

In the first place, it will be seen that they all agree in giving very slight or no growth at all on gelatine at room temperature. Another resemblance is that they all produce acid in litmus milk after a considerable interval of time. Three of them, however, produce acid and clot in litmus milk in forty-eight hours. These are No. 20, Table I.; No. 2, Table XII.; and No. 9, Table XIII.

Again, a large number of them produce a trace of Indol in peptone water, all of them produce a trace of nitrites in nitrate water, and a large number of them produce acid (some of them acid and gas) in cane sugar. The general resemblance in their reactions between organisms 1, 2, 3, 4, and 5, 14 and 15, of Table I., derived from trachea, lungs and glands, and heart-blood of pig 1, and organisms 1, 2, 3, 4, 5, 6, 7, 8 of

Table XIII., derived from the intestines of several small pigs, may be here also pointed out. These are some of the resemblances. The tables have, however, only to be glanced at to appreciate the differences, such as one organism producing acid and gas in a sugar in which another produces acid only, or one organism producing acid in a sugar in which another produces no change. It would take up too much space to go into all the details and attempt to reduce them to harmony. It will suffice if attention is again directed to the discussion on organisms Nos. 14-21, Table I. (pp. 5 and 7), and Nos. 1-8, Table XIII. (pp. 39, 40, 41), where the variations in cultures derived from single colonies of an original pure culture from each other and from the original culture are gone into. In fact, to state the matter briefly, this great capacity for variation in the biological reactions, as also a similar capacity for variation in morphological appearances, constitutes, in reality, one of the greatest resemblances among the organisms we have just been dealing with.

Taking into consideration, therefore, the nature of the disease considered pathologically,—a hæmorrhagic septicæmia; the sources of this organism in cases of the disease; and the practically pure state in which it is in most cases obtained from these sources; its morphological and cultural resemblances; and its further marked capacity to vary morphologically and culturally,—the presumption in my view is that we are dealing with one organism only in all these cases—namely, the *B. suis* *septicus*.

EFFECT OF VACCINATION.

It has already been stated (*vide supra*) that certain of the pigs were vaccinated with a vaccine prepared in a special manner, immediately on their admission to the piggery in April and May. To elucidate the effect of this vaccination, the pigs brought in at that time, 73 in number, will be divided into two classes as follows:—

Class 1, consisting of pigs of groups γ and δ , made up of pigs over 4 months old, and consisting of 36 animals.

Class 2, consisting of pigs of group β , made up of pigs of about 2 months old, and consisting of 37 animals.

CLASS 1.

Number of pigs.	Source.	Vaccinated.	Unvaccinated.
13	A	9	4
8 (pigs)	D	4	4
6 (sows)	D	6	0
9	B	9	0
Thus of 36 pigs, sources A, D, and B, 28 were vaccinated and 8 unvaccinated.			

The following is the number of animals dead and alive of each lot from the various sources at end of August:—

Source.	Vaccinated (dead).	Unvaccinated (dead).	Leaving alive	
			Vaccinated.	Unvaccinated.
A	7	4	2	0
D (pigs)	0	3	4	1
D (sows)	1	0	5	0
B	6	0	3	0
Total . .	14	7	14	1

But as there were no controls in the case of the sows from source D and the pigs from source B, a better indication of the result of the vaccination may be obtained by considering only the pigs from source A and the pigs from source D in the above table.

Thus 7 vaccinated and 7 unvaccinated died, leaving 6 vaccinated and 1 unvaccinated alive out of 13 and 8 respectively.

Again, in the above tables, several pigs were killed, not because they were really ill, but as they were in fair condition to allow of their carcasses being made use of. Hence tables giving, 1st, the results as moribund and dead pigs among vaccinated and unvaccinated; and 2nd, the results

in regard to intestinal lesions among vaccinated and unvaccinated, might give a fairer idea of the result of vaccination.

1st, *Moribund and dead pigs*.—When all the 36 pigs are considered, it is found that there are 3 moribund and dead pigs out of 28 vaccinated, and 2 moribund and dead pigs out of 8 unvaccinated; but if we limit the consideration to the sets of pigs where there were controls (pigs, sources A and D), then there are 2 moribund and dead pigs out of 9 vaccinated, and 2 moribund and dead pigs out of 4 unvaccinated.

2nd, *Intestinal lesions*.—Here the whole 36 pigs will be considered, and the result is that 2 out of 28 vaccinated and 4 out of 8 unvaccinated showed these lesions.

It will thus be seen that, so far as any deduction can be drawn from the results, the unvaccinated suffered more severely than the vaccinated.

CLASS 2.

Originally there were 37 pigs in this class, and of these 22 were vaccinated, with 18 dead at end of August; 11 were unvaccinated, with 1 dead at end of August.

(Four pigs of this group were not taken into consideration because they were obviously undersized, thriftless “piners,” to which to give a dose of vaccine was regarded as dangerous, in that it might have killed them. Of these four, two were dead by the end of August.)

There was thus a very much higher death-rate among the vaccinated pigs (18 out of 22) than among the unvaccinated (1 out of 11). It has to be further noted that the unvaccinated took a much milder type of the disease, rapidly got over it, and were soon about twice the size of their vaccinated fellows.

Practically all the pigs of this class at the post-mortem showed well-marked lesions either of the intestine or lung. Considering, however, moribund pigs as actually dead, there were among the vaccinated 13 out of 22, and among the unvaccinated 1 out of 11, again showing the marked preponderance among the vaccinated.

The results, therefore, of the vaccination as performed above, so far as they go, would appear to be possibly favour-

able for the pigs over 4 months, and very unfavourable in the pigs between 2 and 4 months. With regard to the results in the pigs over 4 months, it cannot, however, be excluded that possibly they would have done much better if they had not been vaccinated at all. This is all the more probable in view of the mild affection of the control unvaccinated pigs between 2 and 4 months old. The reason for the high death-rate in the vaccinated pigs between 2 and 4 months may have been that in this latter case too large a dose of vaccine was given for the body weight of the animal, and, as a matter of fact, proportionately to their weight they were given a much larger dose than the older pigs of Class 1. On the other hand, the pigs of Class 2 were obviously not in good health, and, as has been already stated, four of them were markedly piners. These latter were not themselves vaccinated, but they belonged to the same group and came from the same source. Further, two pigs of this class—pigs 9 and 10—were killed twelve days after they came into the piggery, and exhibited lesions which must have long antedated their arrival in the piggery. The question arises, therefore, whether they would not be much more sensitive to the vaccine than the older pigs, which by their very age were more remote from the period of acute lesions. As the pigs of both classes which first took the disease became acutely ill two to three days after the second injection, the question of anaphylaxis is also involved. As, however, the vaccine used was absolutely sterile, something more than anaphylaxis was required to set up the specific disease which actually appeared. The whole question of the mode of action of the so-called “filter passer” is thus raised in an acute form, but as this has already been dealt with at some length in my former paper, it will not be further discussed here.

SOURCE OF INFECTION IN THE TWO OUTBREAKS IN PIGGERY II.

1st outbreak.—Swine fever had broken out in the piggery a year previous to this outbreak. The pigs were then killed out, the place disinfected, and a new stock brought in. Things

had gone well with the pigs during the year, but about the end of February 1916 there was a long spell of very cold, stormy, and snowy weather, and it was while this lasted that the outbreak occurred. There had been no introduction of fresh pigs into the piggery previous to the outbreak, but diarrhoea had been noticed among the small pigs for some considerable time. The first case of swine fever noticed was of the fulminant variety, and occurred in an old brood sow, nursing young. Thereafter a large number of the pigs were noticed to be ill, some of them very acutely so. Although some of these latter gave evidence post-mortem of the acute nature of the disease by the fact that the only post-mortem changes found were multiple hæmorrhages all over the body, several of them exhibited lesions *the initiation of which must have long antedated the discovery of the disease in the piggery*. The presence of the distemper organism in one case from this outbreak should be noted in relation to it being a possible cause of some of the illnesses of pigs classed as swine fever.

The animals were all killed out, and the place thoroughly disinfected with carbolic acid and limewash. A period of 1½ months was now allowed to elapse before fresh pigs were brought in. Having regard to the subsequent history in this piggery, it is important to note that these precautions were taken, and especially to note that the disinfection was very thorough indeed. In view, however, of the opinion expressed by the Departmental Committee on swine fever on page ii. of their final Report in 1915, where it is stated "that a period of fourteen days may be regarded as sufficient to bring about the disinfection of infected manure through natural causes," these precautions would appear to have been excessive. They erred, however, on the safe side; and having been carried out, and that in a very efficient manner, they give little support to the view (which may be advanced) that the disease (which afterwards broke out again in this piggery) was introduced among the fresh pigs brought in subsequently by infection from this previous epidemic.

Fresh pigs began again to be introduced into this piggery about the middle of April, a month and a half nearly after

the place had been cleared out. The pigs introduced came from four sources; and it needs to be emphatically stated *that swine fever was not known to exist in any of these sources by the Board of Agriculture officials or by the vendors or buyers of the pigs.* The pigs were of three ages, as has already been mentioned, group β containing pigs from 2-4 months, group γ from 4-6 months, and group δ above 6 months; and it has to be very specially noted, 1st, that the thirty-seven pigs of group β , which came all from one source, were on the average much undersized and very unthrifty in appearance, four of them being notably so; 2nd, that two pigs of group β (pigs 9 and 10) which were killed on March 22, twelve days after their arrival in the piggery, exhibited lesions in their lungs of such a nature that, allowing for a period for infection to have taken place in the piggery, these lesions could not have developed while the pigs were in this piggery; 3rd, that one of the large pigs from source B, which never during its sojourn in this piggery showed any signs of illness, when it was killed by the butcher as fat on June 15, a month after its arrival in the piggery, showed extensive but quiescent adherent pericarditis and pleurisy. Had this condition originated in this piggery, the animal would almost certainly have given evidence of it. The inference is that this animal previous to coming to this piggery suffered from a condition suspiciously like swine fever in one of its manifestations.

As has already been stated, certain of these pigs were vaccinated in the manner already described. The following events supervened on this vaccination:—

Pig 6, of group γ and from source A, an apparently healthy pig, in a pen with three other apparently healthy pigs of its own age and from the same source, on receiving a second inoculation on May 3, following a first inoculation on April 18, became acutely ill on May 6, and died on May 14. *Post-mortem.*—Although no ulcers were found in the intestine, the condition present was that of acute swine fever.

One of its pen-mates took acutely ill on May 15 with high temperature. It was killed on the 17th, and there was found present enlargement and congestion of the lymphatic glands and some inflammatory congestion of the

intestines. It should especially be noted that there was no pancreas-like condition in either of the lungs. On May 20 the other two pen-mates became ill, but they were better in about two days. They were afterwards killed as fat, and post-mortem showed no evidence of disease.

A second set of happenings occurred among the pigs of group β . Here again two pigs—pigs 9 and 10—of group β and from same source, and in a pen along with pigs 11, 12, 13, and 14, on receiving a second inoculation on May 17, subsequent to a first inoculation on May 10, became very ill on May 21, and were killed on May 22. The condition found post-mortem was obviously an acute attack on the top of something chronic, for the inferior borders of the lungs, the middle lobe on the right side and the postcardial lobe, in both cases were found in the pancreas-like condition described above. It was also noted that pig 10 was a very small undersized pig, and very thin. Later, on June 2 and 16, the pen-mates of these two pigs—pigs 11-14—were killed, because they were *in extremis*.

Meantime the other pigs of this group were seized with the disease, but the lot just described were the first to be demonstrably ill, and they were all killed out by the disease before deaths began to occur among the others.

From the detailed statement just made the following points arise for consideration. First, with regard to pig 6. It became demonstrably ill on May 6, three days after its second inoculation, and died with all the appearances post-mortem of swine fever. It infected by contagion its pen-mates with a very acute disease. One of these died, the only demonstrable post-mortem appearances being congestion of the intestines and lymphatic glands, while the others recovered quickly. The other vaccinated and unvaccinated pigs of this group did not begin to show illness till the disease had become quite general in the piggery (about the middle of June), and, further, these pigs were in a pen by themselves quite away from contact with the small pigs of group β , in which the disease next started.

With regard to pigs 9 and 10 the following points require consideration. They were brought into the piggery on May 10; received their second injection May 17; on May 21 were

demonstrably ill. They were killed on May 22, and showed lesions too long-standing to have been acquired during their sojourn in the piggery. Pig 10, moreover, was noted to be stunted and emaciated. Both pigs belonged to a group whose members on the average were undersized, thin, and unthrifty. They gave rise to a contagious disease which passed first to their pen-mates and then became general. They had no association with the pig 6 and its pen-mates.

The weather during the initiation of the outbreak was quite mild.

With these facts before us, can we arrive at any idea of the source of origin of the disease in these cases?

To my mind, infection from the previous epidemic in this piggery is excluded by—

- (1) The precautions taken with regard to disinfection, and the interval allowed to elapse before new pigs were brought in.
- (2) The distemper organism, the cause of a very infectious disease amongst a large variety of other animals, was found in pure extensive culture as the only organism present in an extensive pneumonic condition in one of the pigs of the first epidemic. Presumably if the disinfection had not been sufficient this disease and the causal organism ought to have appeared in the second epidemic, but though carefully examined for it was never found.

In the next place, the disease did not arise as such, by being brought in¹ with the fresh pigs from their various sources, because the disease was not known to exist in these sources, and vendor and buyer were satisfied with regard to this point.

To my mind the disease arose in the piggery by the administration of the vaccine to pigs suffering from latent and unrecognised lesions of the disease, and for the following reasons:—

- (1) The facts already adduced (*vide supra*) anent the clinical and pathological condition of the pigs when they arrived at the piggery;

¹ In a patent form that is. To my mind it was brought in, however, in its latent unrecognised form.

- (2) The undoubted relation of the appearance of the first demonstrable cases to the second administration of the vaccine, especially in the light of the well-known phenomena of anaphylaxis ;
- (3) The difference of behaviour of the vaccinated and unvaccinated lots of group β , as already described, undoubtedly points again to the administration of the vaccine being in some way a causal factor.

It would be wise at this point to clear up what may appear at first sight to be a contradiction in terms. It might seem that if it is true that the pigs were suffering from latent and unrecognised lesions of the disease when they arrived at this piggery, then they must have been suffering from the disease when they were at the sources from which they were derived. In this connection there has first to be borne in mind the fact that the disease was not known to be present, officially or otherwise, at these sources; next, the reputed difficulties in connection with the diagnosis of swine fever in certain of its manifestations; and further, that the diagnosis when it is made, depending as it does in the last instance on the finding of ulcers in the intestine, leaves many loopholes for cases of the disease being missed. In the first place, as long as pigs are going on fairly well in a piggery, and there are no sudden, acute deaths taking place among the larger pigs, the owner has no reason to suspect the presence of swine fever. Small suckling pigs may be suffering and dying from diarrhoea, and may be becoming piners and wasters with a chronic cough, and unthrifty to the last degree. Such happenings, though recognised by pig-keepers as occurring, and occurring very frequently, in pigs, are unassociated in their minds with swine fever. Suspicion is first aroused, however, when a large number of the largest and most valuable pigs die suddenly with all the symptoms of a very acute illness. The process thereafter consists in a search throughout the piggery for an animal to show the so-called characteristic ulcers of the disease. When this is found the condition is stamped swine fever. *It is not recognised, however, that these lesions when found must have long antedated the sudden outburst which drew attention to the disease, nor is it recognised that had it not been for*

this sudden outburst, the possibility is that these pigs with the ulcers in their intestines would have lived, and been killed eventually and sold as pork without anybody being the wiser. (These considerations are brought forward without imputing anything but the most proper motives in all concerned.)

A further point which has also to be borne in mind in regard to the difficulty of recognising when an animal is really ill is the disparity between the gravity of the lesions with which many pigs (and other lower animals) can be affected and the symptoms exhibited. Thus I have seen puppies, whose lungs were almost completely solidified with the pneumonic condition so commonly found in distemper, jumping and frisking about in a manner quite unexpected considering the gravity of their condition.

Returning again to the point as to what may be termed latent and unrecognised lesions of the disease, the view is commonly held that pneumonic conditions and chronic pneumonia have nothing whatever to do with swine fever. They are looked on as mere accidents in the disease, and as being the result, according to the prevalent view, of secondary infections, having no relation whatever to the primary cause of the disease. In this connection I can only point to the constancy with which cough occurred as a symptom in the outbreak here investigated, with which pneumonia was found in the various cases and with which *B. suis* was isolated in pure extensive culture from this lesion; to the absence of other bacterial causes of pneumonia; and finally, to the complete cycle which I believe I have established in this outbreak, symptomatologically, pathologically, and bacteriologically.

In connection with the further study of the epidemiology of the disease in this outbreak, the phenomena observed in connection with the six sows obtained from source D requires further consideration. These sows were in young when they arrived in the piggery about the beginning of May 1916. When they first came they were kept in the large stationary piggery with which we have been dealing up to the present. They were vaccinated twice, first on May 3 and then on May 10. After the second vaccination

they were at once removed to a new wooden piggery at some distance from the original one. This piggery was entirely new, and was situated on ground where no pigs had been before. It was run as an entirely separate establishment with a separate staff, there being no communication between the two places. The sows remained apparently well till about the end of June, when four of them showed signs of an acute illness of a more or less transient nature, and all got better. They began to farrow about the 20th of July; thereafter the two sows which had not been ill previously became acutely and severely ill with symptoms of a hæmorrhagic colitis, and one of them died. The pigs from all these sows when they were about a fortnight old began to be affected acutely with diarrhœa. This diarrhœa was associated at first solely with an acute superficial hæmorrhagic and necrotic inflammation of the large intestine. As the pigs grew older definite ulcers of the large intestine appeared, and later still definite pneumonia, hæmorrhages, effusion into the pericardial sac, and definite pericarditis occurred. At the same time the symptomatology shifted in accordance with the lesions. Bacteriologically, the *B. suis* *septicus* was found at first chiefly in the large intestine, but later its appearance was signalled in the heart-blood, pneumonia, and lymphatic gland condition. So that from the simple diarrhœa of these sucklings there was evolved in the course of a month and a half the symptomatology, pathology, and bacteriological findings of the small pigs of group β , with which the epidemic started in May. These suckling pigs must have got the disease from their mothers, as they were near no other pigs, and their mothers undoubtedly were very acutely ill of a disease which resembled somewhat the type of affection in the case of the pen-mates of pig 6 and somewhat that of pig 27. How the mothers obtained the infection is doubtful. The disease occurred among them too late, and was of too acute a type for it to be likely that it was related to the vaccination or to infection while they were staying in the original piggery. It is just possible that in this instance the infection was carried on the feet of birds. The new piggery was in a wood, and was about 150 yards from the old one, and large

flocks of birds, especially starlings, fed in the troughs of both places.

It would be well, in closing this section, to draw attention again to the importance attached here from the point of view of the spreading of the disease, to diarrhoea in small pigs, and the syndrome mentioned above, consisting of discharge from the nose and eyes, and special skin eruption, being recognised as definite items in swine fever; and to the presence of cough, spontaneous or appearing when the animals are hustled, as a very suspicious indication of the same disease in a latent form, if helminthiasis of the lung is excluded.

It should also be noted that the view is put forward here, that in all probability the disease swine fever is more or less, so to speak, always with us, requiring only the subjection of the animals suddenly to intense inimical conditions for its appearance in the form in which it arouses suspicion of its existence—namely, by attacking fulminantly and fatally several of the larger and more valuable animals. The state of matters suggested as existing here may be illustrated by reference to Plant Pathology. Potato disease due to *Phytophthora infestans* is always more or less present in the form of a diseased tuber here and there in possibly every field in the country. It takes, however, a wet sunless summer, such as occurred in 1916, to convert the potential danger of a few diseased potatoes into a real one in the shape of the failure of the entire potato crop in the country through potato disease.

SUMMARY AND CONCLUSIONS.

Symptomatology.

Special attention is directed to the practical constancy of the diarrhoea in the young pigs of suckling age, and its absence as a rule in pigs over this age, where simple constipation or constipation of the type described in the text was the rule; to the existence of the syndrome, as described; to the existence of cough, especially among the older pigs, as a chronic latent manifestation of the disease; to the type of

paralysis exhibited in this epidemic as described; to the very indefinite nature of the symptoms exhibited in many cases, possibly associated with the fact that the disease occurred during the summer; and to the various skin eruptions observed in the outbreak.

Pathology.

Here the notable facts are—the presence in very young suckling pigs of a superficial necrotic hæmorrhagic inflammation, especially of the large intestine, associated with the diarrhoea just mentioned; as these pigs get slightly older, the addition to this lesion, of discrete ulcers in the intestine, acute pneumonic changes in the lung, together with pericarditis; in older pigs still, the gradual disappearance and resolution of these lesions, although it should be stated that up to the age of eight months the pneumonic condition in the lung still persisted; the important rôle attributed here to hæmorrhage in this disease in that it is held to explain the majority of the lesions observed, and especially the “button ulcers” of the intestine; the changes in the anterior horn of the grey matter of the spinal cord in the case of paralysis associated with the presence of the *distemper bacillus* should be noted in passing.

Bacteriology.

Here attention may be drawn to—

The comparative rarity with which *B. suispestifer* of the classical type was isolated from these cases; the fact that the *distemper bacillus* was obtained from the pneumonic lung in pure extensive culture in one case and mixed with *B. suissepticus* in another case of supposed swine fever; its absence from the cases in the large epidemic in Piggery II. should, however, be noted at the same time.

The frequency with which *B. suissepticus* was obtained in pure extensive culture from various situations in the body in all the epidemics. Especially should it be

noted how the number of sources extended, from which the organism was obtainable, as the suckling pigs of group *a* grew older and developed other lesions.

Immunity and Vaccination.

The questionable favourable result in the older pigs obtained from vaccination, and the very unfavourable results in the younger pigs, together with the possible explanations of these results, as given in the text, may be here mentioned.

Epidemiology.

The view is expressed here that swine fever is more or less endemic, under present conditions, in a suppressed or latent form wherever numbers of pigs are kept together. The advent of grave inimical conditions would appear to light up this latent condition, causing acutely fatal cases which leads to its discovery and diagnosis as swine fever.

It is here suggested, that to recognise the latent manifestations of the disease attention should be paid to the diarrhoea in sucklings, to the presence of piners or wasters dating from the suckling age, to the existence of the syndrome mentioned above, to the presence of cough and unthriftiness among the older pigs, and to a less extent to the presence of a rise of temperature.

In conclusion, the facts derived from a consideration of these outbreaks would appear to support the conclusions drawn in my previous paper on this subject, and which have been mentioned above in the introduction.

RECOMMENDATIONS.

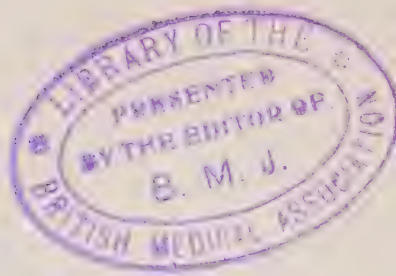
- (1) The recognition of latent and hitherto unrecognised manifestations of the disease.
- (2) Dealing in the appropriate manner with animals affected with these latent and unrecognised manifestations of the disease.

- (3) Attending to the general health of the pigs, and, especially, protecting them from grave inimical conditions such as sudden exposure to extreme cold, especially when this is associated with rain or snow.

ACKNOWLEDGMENTS.

I desire to express my very great indebtedness to the Managers of the Royal Edinburgh Asylum and to Dr Robertson, superintendent thereof, for the great facilities afforded me during the investigation. To Mr John Mac-Intosh and Mr Peter Elmslie of the above Institution I am also much obliged for help cordially given. For suggestions and corrections during the progress of the work I have to thank Professor James Ritchie, Mr M'Callum, Director of the Edinburgh and East of Scotland College of Agriculture, and Mr Barber of the Board of Agriculture for Scotland. I am also specially indebted to Mr M'Callum for seeing this article through the Press.

[ILLUSTRATIONS.



ILLUSTRATIONS.

FIG.

1. Small pig of group β , showing discharge from eyes.
2. Small pig of group β , showing macular dark hæmorrhagic rash all over body.
3. Heart of small pig of group β , showing extensive subepicardial hæmorrhage.
4. Bladder from pig of group β , showing (*a*) the hæmorrhages.
5. Section of bladder of pig 4, showing (*a*) the mucous membrane, (*b*) the hæmorrhages, and (*c*) the muscle. Note that the hæmorrhage comes right up against the mucous membrane, and that in places the red blood cells are penetrating between the cells of the epithelium.
6. Section of part of kidney from pig of group β , showing (*a*) the pelvis of the kidney, (*b*) a papilla cut through, and (*c*) part of the hæmorrhage.
7. Section of lung from pig of group β , showing (*a*) hæmorrhages, and (*b*) catarrhal broncho-pneumonia.
8. Section of wall of large intestine, showing (*a*) large hæmorrhages in the submucosa. The mucous membrane is bulged into the lumen of the gut owing to the size of the hæmorrhage.
9. Section of lymphatic gland, showing (*a*) the hæmorrhages, (*b*) lymphoid tissue.
10. Section of necrotic ulcer on nose of pig, showing (*a*) necrotic swollen epithelium, (*b*) junction of swollen epithelium, with normal epithelium, (*c*) normal epithelium, (*d*) line of demarcation between normal and necrotic tissue.
11. Same as in fig. 10, under higher magnification: lettering the same.
12. Necrotic ulcer on the tip of the tongue (pig 41), showing (*a*) necrosed portion, (*b*) line of demarcation between necrosed and healthy tissue, (*c*) organising thrombi in subjacent blood-vessels.

13. Some coils of large intestine of suckling pig, aged about one month, and affected with diarrhoea, showing (a) healthy mucous membrane, (b) two ulcers, (c) extensive diphtheritic exudate on the mucosa of rest of large intestine.
14. Section of the portion of this intestine with the diphtheritic exudate, showing (a) necrosis of superficial layers of mucous membrane and remains of necrosed gland tubules, (there is also hæmorrhage into this layer), (b) more healthy layer of mucous membrane, showing, however, at (c) hæmorrhages, and great engorgement of the vessels with blood, (d) submucous layer, (e) muscular layer, (f) serous layer, showing great œdema.
15. Necrotic ulcer in large intestine, showing (a) normal mucosa, (b) junction between normal and necrotic mucosa, (c) necrotic mucosa, (d) swollen submucous tissue, (e) muscle of intestinal wall, (f) line of demarcation between necrosed and living tissue.
16. More advanced necrotic ulcer, showing (a) healthy mucous membrane, (b) necrosed mucous membrane, (c) collections of blood undergoing necrobiosis, (d) line of demarcation between necrosed and living tissue, (e) organising thrombi in subjacent blood-vessels, (f) muscular wall of intestines, (g) ulcer penetrating through to serous covering.
17. Another of the same, letter references as in fig. 16.
18. Blood-vessels of large intestine as seen in vertical section (from Schäfer after Kölliker), showing (a) an artery passing up from submucosa, (c) vein arising from capillary plexus (b) which surrounds the mouths of the glands.
19. Heart of small pig of group a, aged about one month, showing pericarditis at (a).
20. Section of lumbar enlargement of pig 4, showing (a) infiltration of blood-vessels with small round cells, (b) chromatolysis of nerve-cells in anterior horn.





Fig 1.



Fig 2.



Fig. 3.

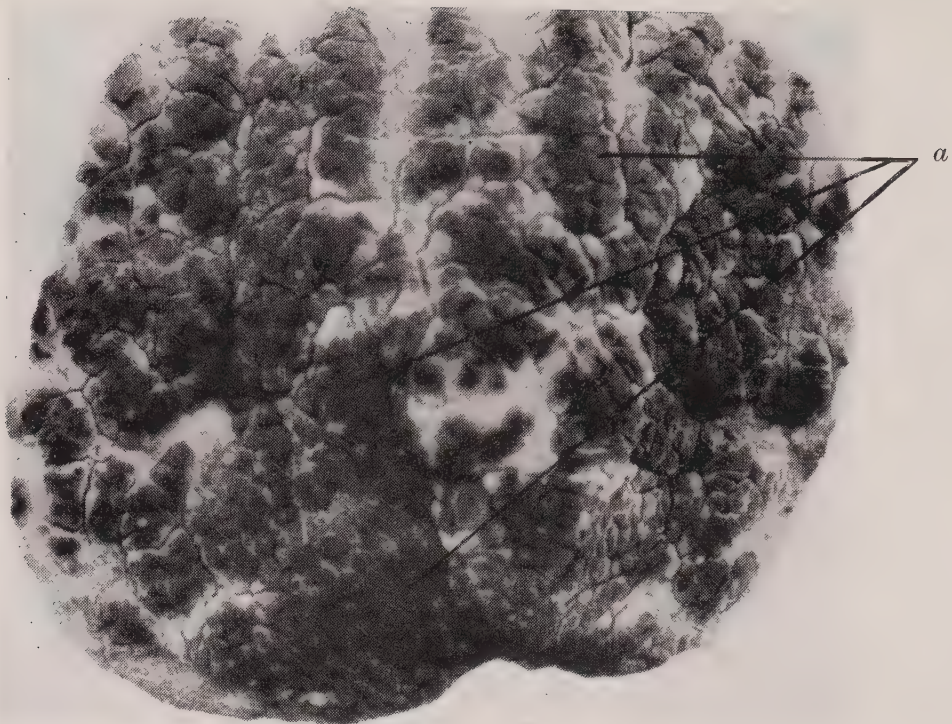


Fig. 4.

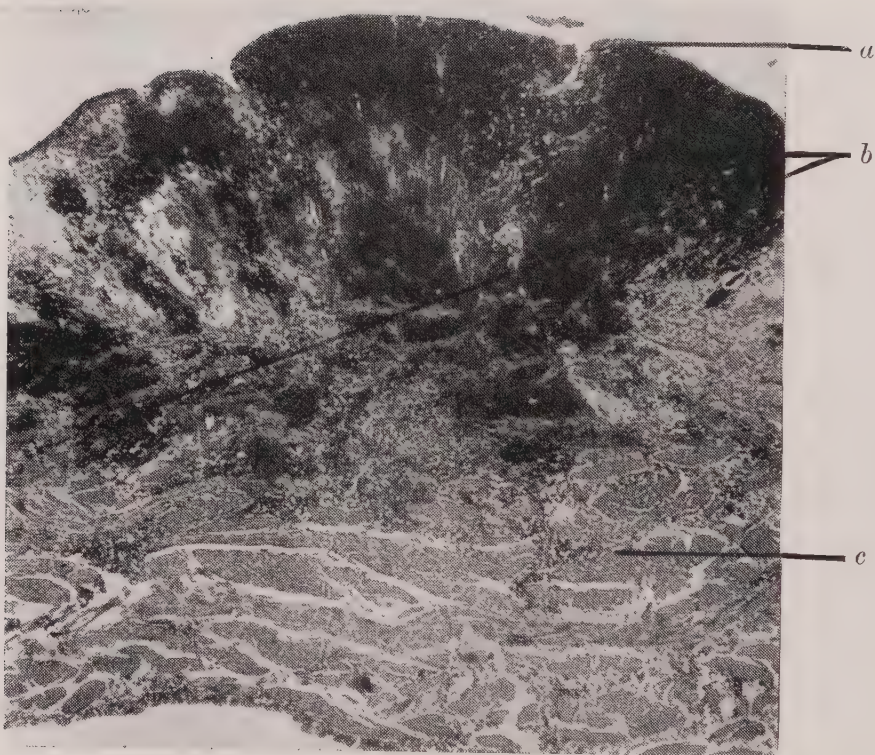


Fig. 5.

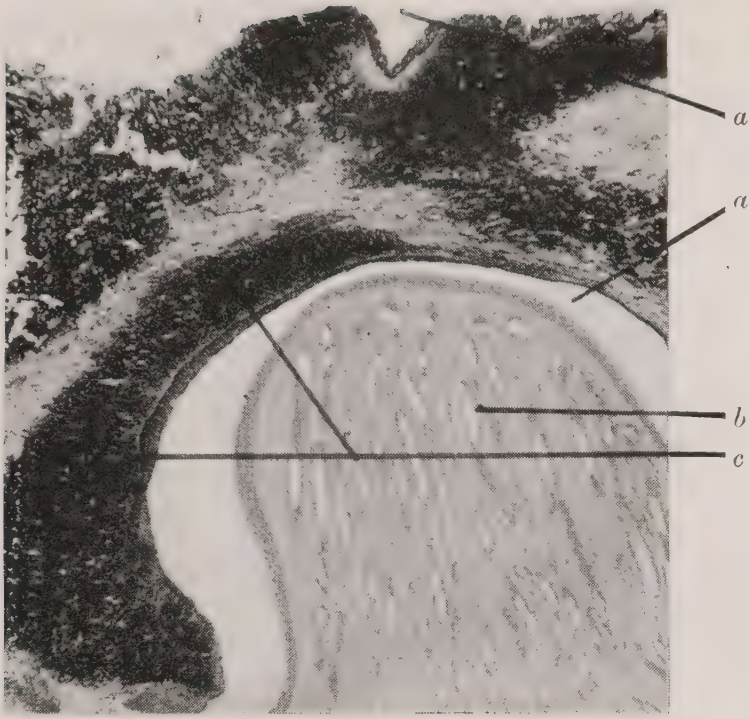


Fig. 6.

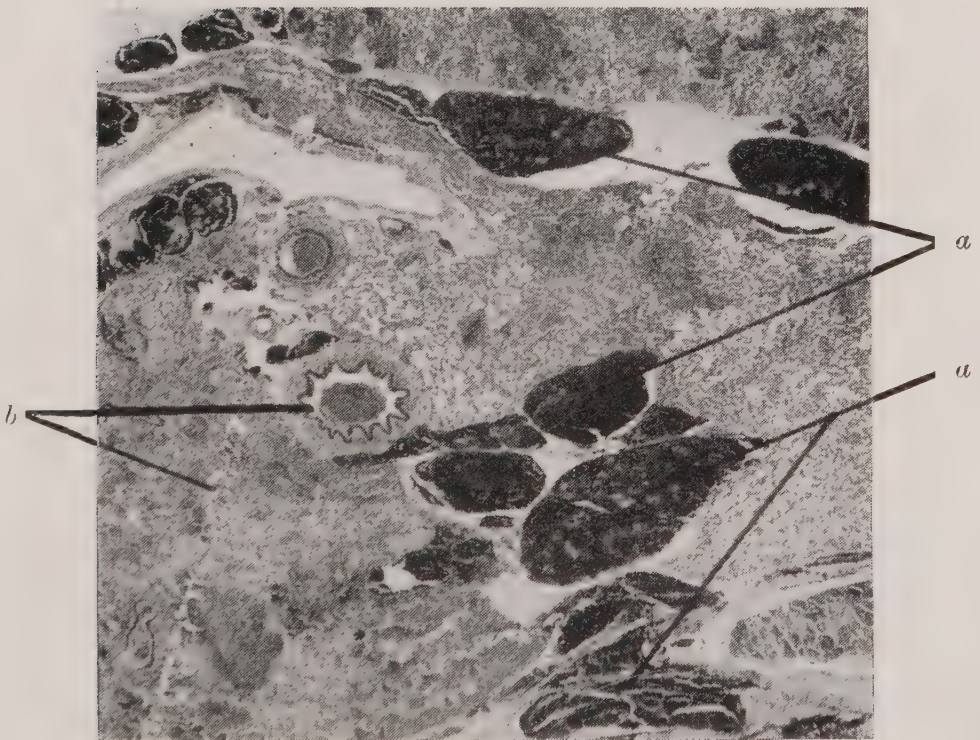


Fig. 7.

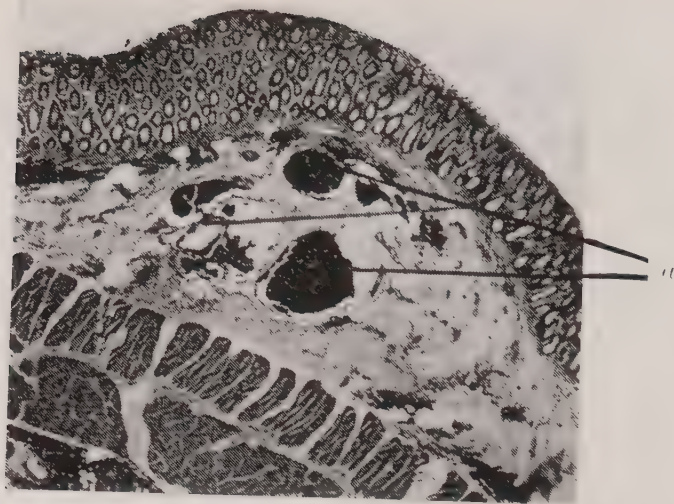


Fig. 8.

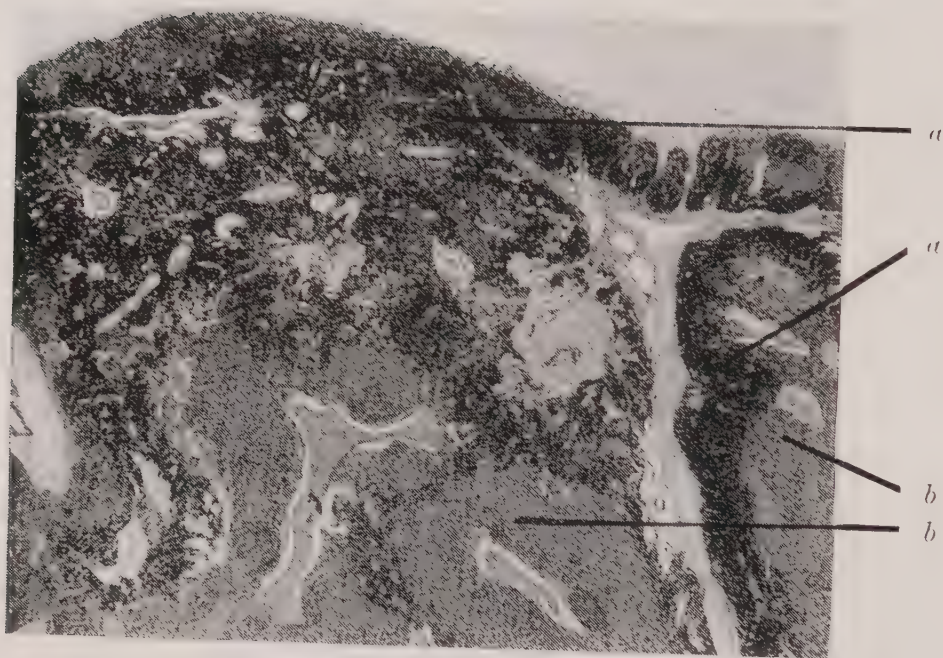


Fig. 9.



Fig. 10.

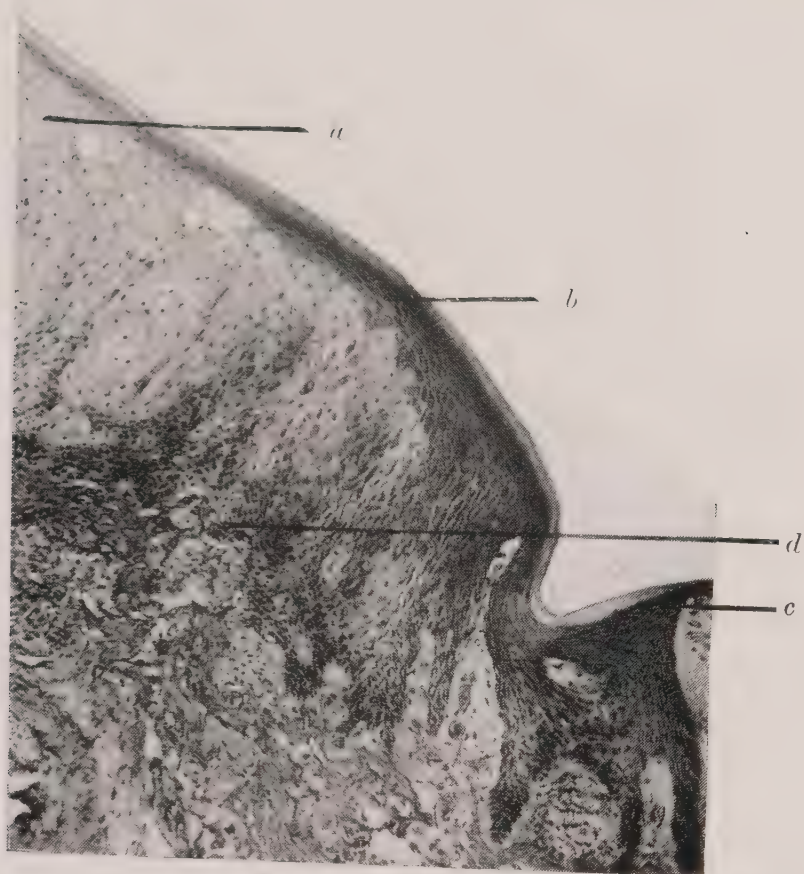


Fig. 11.



Fig. 12.

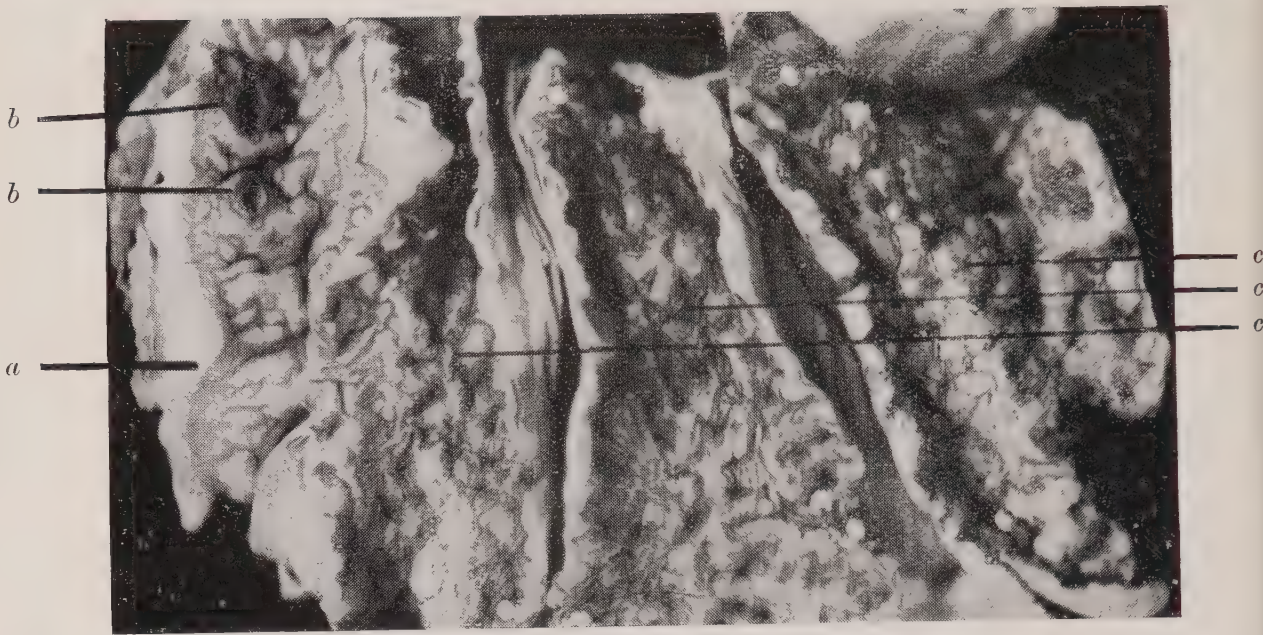


Fig. 13.



Fig. 14.



Fig. 15.

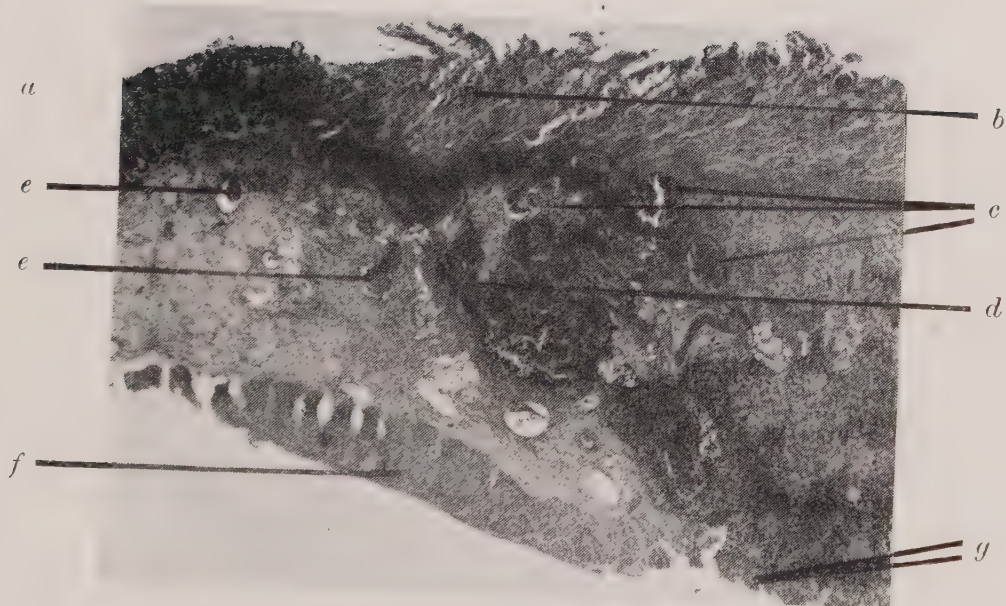


Fig. 16.

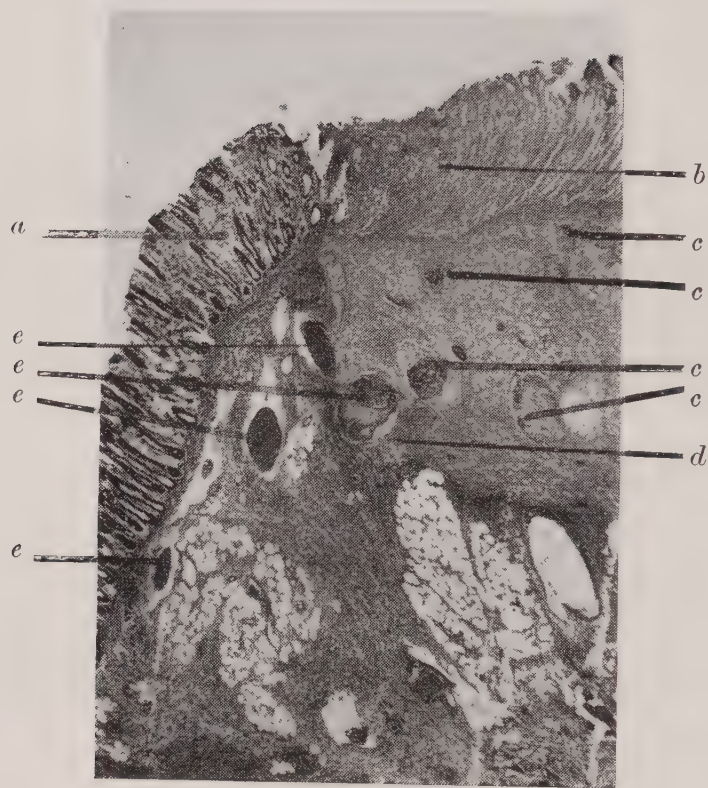


Fig. 17.

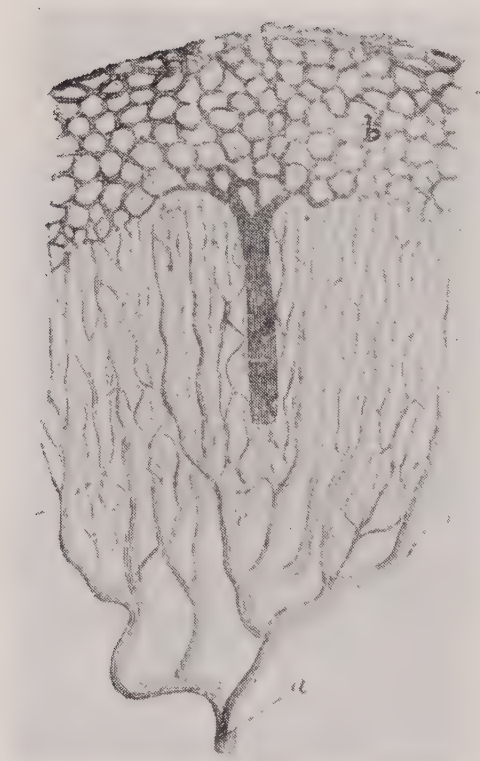


Fig. 18.

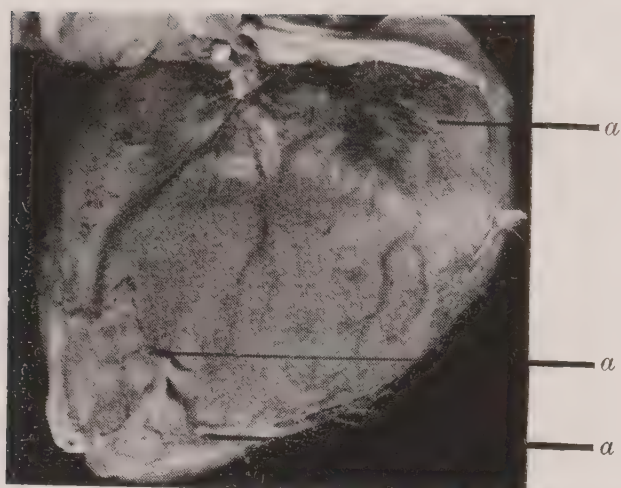


Fig. 19.

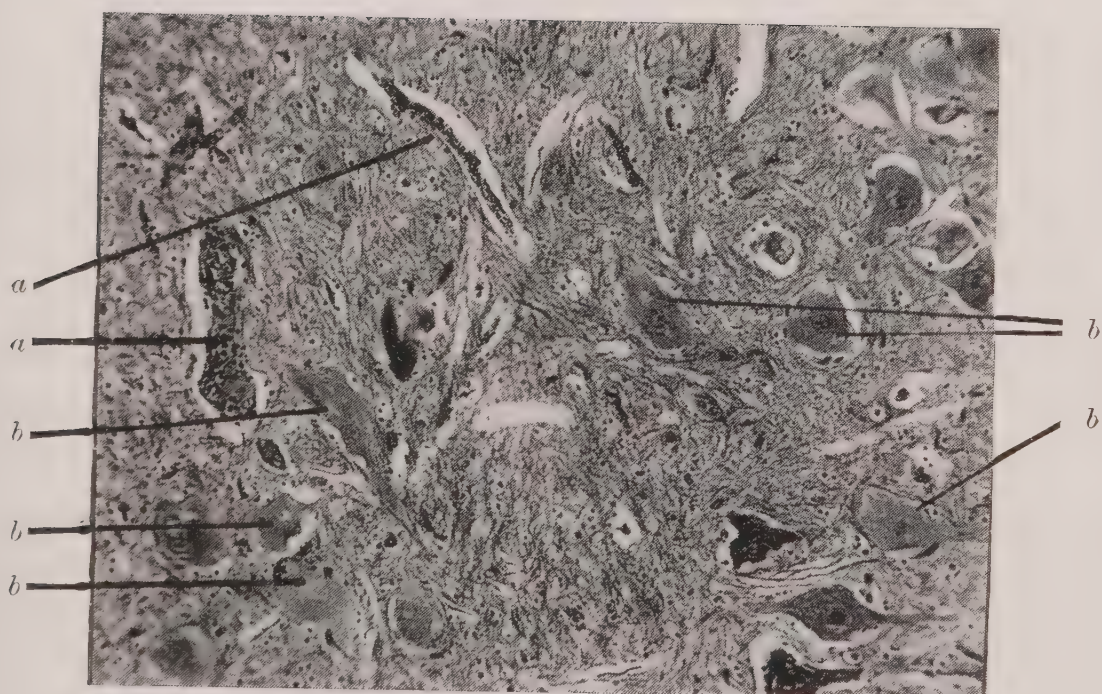


Fig. 20.

